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# THE CUTANEOUS MANIFESTATIONS OF POLYARTERITIS NODOSA.

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The symptoms and signs of polyarteritis nodosa have become sufficiently well recognized in recent years for a diagnosis to be made on clinical grounds. Skin lesions in the disease have attracted attention mainly in foreign literature: Alkiewicz (1933) collected 150 cases from the literature of 1866 onwards and found the skin affected in 36 of these; Ketron and Bernstein (1939) estimated the incidence of skin affection as 25% of all cases. Skin manifestations may therefore be of significant help in diagnosis when found in conjunction with other evidence of the disease.

A sufficient number of cases of polyarteritis nodosa has now been described for us to appreciate the types of eruption commonly seen. The acute stage of the disease is accompanied by polymorphic exanthemata but the more characteristic manifestations reflect the vascular damage present, haemorrhage and gangrene being found in the acute stages, racemose livedo and ulceration in the chronic phase; subcutaneous nodules, being the essential lesion, may occur at any stage of activity.

*Nodules.*—Nodules in the skin and subcutaneous tissues were described in the original cases of Kussmaul and Maier (1866). The picture they presented of an acute febrile illness with pallor, weakness, extreme muscle pains, colic, paraesthesiae, tachycardia, haematuria and albuminuria with a short course ending always in death remained the whole conception of the disease for many decades. Of recent years, however, many cases have been described in which

the diagnosis has been confirmed by biopsy of a subcutaneous nodule where the patient subsequently recovered. Indeed Lindberg (1931, 1932), describing two cases of four and nine years' duration who had subcutaneous nodules and no systemic signs, postulated a cutaneous form with a relatively good prognosis.

Lamb (1914) considered that subcutaneous nodules offer the only certain means of diagnosis during life, yet they are found in only 20% of cases (Miller, 1949). When nodules are present they may number up to fifty and show a tendency to grouping, giving the impression of shotty thickenings along the course of an artery, and varying in size from that of a pea to a hazel nut (Keil, 1938). They tend to appear in crops and may regress in a few days or persist for periods up to eighteen months (Miller and Daley, 1946). The lesions are sometimes painful and tender and have been observed to pulsate (Manges and Baehr, 1921 ; Spiegel, 1936). The overlying skin may be unchanged, reddened, thinned and glistening, or may ulcerate (Vining, 1938).

The resemblance of the lesions to erythema nodosum has been remarked upon in several cases (Klotz, 1917 ; Spiegel, 1936 ; Vining, 1938 ; Miescher, 1946) and although the histological appearances may lead to confusion, since the primary lesion in both diseases is a vasculitis, the clinical course is sufficiently dissimilar for differentiation.

*Ecchymosis and gangrene.*—Severe subcutaneous haemorrhages resulting from rupture of an aneurysm or weakened arterial wall are a characteristic, though not common, feature of the acute stage. Typically they are symmetrical and are followed by gangrene.

Taylor and Jacoby (1949) record a case of multiple subcutaneous haemorrhages in a child of seven, and draw an analogy between the haemorrhagic eruptions of polyarteritis nodosa, purpura fulminans, post-scarlatinal purpura and gangrene, and purpura necrotica, postulating a common aetiology. Similar cases of severe symmetrical subcutaneous haemorrhage which resulted in gangrene of the affected areas have been reported (Frommel, 1926 ; Freund, 1926 ; Melczer and Venkei, 1947). Symmetrical cutaneous gangrene without preceding haemorrhage occurred in a case reported by Löhe and Rosenfeld (1931). The lesions commenced as raised urticarial patches many of which became discoloured and ultimately gangrenous. The ears, cheeks, arms and areas on the trunk were affected ; they healed in about a month and the patient recovered. Miller and Daley (1946) also record a similar patient who recovered.

Gangrene of the extremities may occur as in the case described by Barnard and Burbury (1934) of an acute symmetrical gangrene in all four limbs following a three weeks' febrile illness in a girl aged eight. Lesions of polyarteritis nodosa were found at autopsy, as they were in other cases of peripheral gangrene recorded by Cohen *et al.* (1936) and Carr (1930).

In the chronic stage, affection of the peripheral vessels may give rise to

Raynaud's phenomenon and a dry gangrene may develop in the extremities (Miller and Daley, 1946 ; Bernstein, 1935).

*Acute exanthemata.*—The acute stage of polyarteritis nodosa is accompanied by various exanthemata of which purpura is the commonest. Thus Spiegel (1936) records 17 cases of acute polyarteritis nodosa, 12 of whom had skin eruptions half of which were purpuric. Alkiewicz (1933) found purpura described in 16 of the 36 cases with skin manifestations recorded at that time. The platelet count may be normal as in Spiegel's cases, but Dunbar (1936) recorded a case with thrombocytopenic purpura. Paradoxically the platelet count may be increased in this disease (Spiegel, 1936). Other cases with a purpuric eruption are reported by Krahulik *et al.* (1935), Motley (1936), Beattie and Douglas (1913) and Bennett and Levine (1929).

Spiegel was impressed by the erythema multiforme type of eruption which occurred in four of her cases, and drew attention to the resemblance between Osler's erythema multiforme with visceral manifestations and polyarteritis nodosa. A papulo-bullous rash with ulceration of the mouth was present in the acute case recorded by Debré *et al.* (1928). A similar vesicular rash was found in one of Grant's (1940) cases, while another of the same series had typical erythema multiforme.

Urticaria is described in the acute stage but is usually accompanied or succeeded by a haemorrhagic eruption (Spiegel, 1936 ; Grant, 1940 ; Cohen *et al.*, 1936).

The relationship of polyarteritis nodosa to a streptococcal infection has frequently been noticed and cases have been recorded after scarlet fever (Spiegel, 1936) and erysipelas (Dietrich, 1933). Scarlatiniform eruptions and erysipelas may also occur during the course of the disease (Freund, 1926 ; Matras, 1938).

The polymorphic character of most of the acute exanthemata described in this disease is well illustrated in the case recorded by Hutinel *et al.* (1930) in which the illness was initially accompanied by a scarlatiniform eruption on the legs. This faded and was followed by erythema multiforme with vesiculation and purpura ; in the terminal stages necrosis and ulceration developed.

*Oedema.*—Oedema is present in about half the recorded cases of polyarteritis nodosa during some stage in its course (Miller, 1949). It may be due to congestive heart failure or may be renal in origin, and with these causes we are not concerned in this paper. Of dermatological interest is the oedema which may arise in association with the polymyositis which occurs in 50% of cases (Boyd and Nussbaum, 1936). The resultant illness, characterized by fever, skin eruptions, muscular pain and weakness and oedema of the face and limbs, may be clinically indistinguishable from dermatomyositis.

*Livedo reticularis.*—Livedo reticularis occurs in some normal subjects as a

transient phenomenon on exposure to cold. Lewis (1927) states that it is due to variation in blood supply in which the pale central areas represent areas of maximal flow. In marginal zones, where flow is less because of lack of tone in the vessels, any impediment to the blood supply—in spasm or inflammatory occlusion—might accentuate and perpetuate this pattern. Erhmann (1907) described 9 cases of reticular livedo in which he postulated a syphilitic origin, with the name of livedo racemosa. Since the Wassermann reaction was not then in general use his evidence rested on the presence of undoubted clinical syphilis in some of the cases. Adamson (1916) invoked a tuberculous aetiology in his series of 6 cases of livedo reticularis. At least one of these (Case 4) bears a close resemblance to the cases of polyarteritis nodosa which we record.

A persistent reticular livedo was first described in polyarteritis nodosa by Goldschlag and Chwalibogowski (1935). Their patient, a boy aged five, developed a dark blue livedo on the limbs and trunk, the larger strands of the pattern being palpable ; this was accompanied by a nodular eruption and a polymorphic rash of urticarial, macular and papular lesions. The diagnosis was confirmed by biopsy of a nodule. Over a period of one year the child had several relapses but at the end of that time he recovered.

The production of livedo reticularis in polyarteritis nodosa is essentially a chronic process and in most described cases showing this manifestation the disease has entered a chronic phase. We can trace only one case in which the eruption occurred in the acute phase and in which the patient died (Ketron and Bernstein, 1939). In this case the patient aged 37 suffered a fulminating attack of the disease with all the accepted systemic signs and died in 7 weeks. During the course of this illness a reticular livedo developed on the trunk and limbs, dark purplish-red in colour with some raised, tender haemorrhagic areas. In indefinitely defined areas on the arms and thighs the skin had a chalky appearance with a slightly thickened feel. Before death the haemorrhagic areas became necrotic and ulcerated.

Dowling (1943) reported the case of a boy aged 15 who developed tender nodules while ill in hospital with " rheumatic fever". The pyrexia continued for a year, red nodules appeared in bouts, being proved by biopsy to consist of the lesions of polyarteritis nodosa, and a blue mottling developed over the greater part of his body. A similar case which had continued in activity since an attack of " rheumatic fever " three and a half years before is recorded by Guequierre and Greenbaum (1942). The original illness had been accompanied by stiffness in the muscles of the legs which persisted.

Oliver (1948) reported a case under the heading of panarteritis which was later thought to be polyarteritis nodosa. The patient, a man aged 19, for three years had recurrent crops of tender subcutaneous nodules and developed livedo reticularis over the trunk and lower limbs. The skin of both legs is described as tense and somewhat indurated.

Similar chronic cases with livedo and nodules are recorded by Crosti (1935), Carol and Prakken (1937), Miller and Daley (1946), Kimberley (1946), Ormsby (1948) and Slinger and Stark (1951).

*Ulceration.*—A case of polyarteritis nodosa with chronic ulceration and livedo reticularis was shown at the Royal Society of Medicine in London at the same time as Case 1 in this report (Barber, 1949). The ulcers developed in a similar way to those in cases with acute skin gangrene, commencing as violet areas of discoloration many of which became necrotic and ulcerated. This process had continued for 18 months.

A similar case is recorded by Matras (1938); the ulceration, which started on the legs, resembled varicose ulceration until it occurred on other areas and livedo reticularis developed.

Under the title of asphyxia reticularis multiplex, Unna (1894) described a similar eruption of three years' duration in a woman aged 40 after an attack of what appeared to be erysipelas.

A recurrent ulcerative eruption was described by Alkiewicz (1933). The lesions commenced as reddish nodules varying from about one to three centimetres in diameter, sometimes with a slightly scaly surface. These became necrotic and ulcerated, ultimately healing leaving an atrophic scar. The crops of nodules were accompanied by a fever and continued until the patient died two years after the onset of the illness.

## Case Reports.

Case 1.—A man aged 22 was admitted to hospital on 3 March 1949 complaining of mottling and ulceration of the skin.

His illness started in 1941 with an attack of universal joint pains which kept him in bed for 6 months. He was well after this until 1944 when he woke one morning quite paralysed and in the next few days his face swelled sufficiently to close his eyes. He suffered transient attacks of agonising pain which made him cry out; the pain was not in the joints but in the muscles. His paralysis and pain lasted for three months. The power returned to the upper part of his body before the lower. He had sweated profusely and his weight had fallen from 12 stone at the beginning to 7 stone 5 pounds at the end of his illness. This second illness kept him in bed for a year. During both attacks he had received sulphonamides in unknown quantity.

The skin changes began insidiously after the second illness: A red mark on one leg ulcerated and then scabbed over, and this process spread over ever wider areas of skin. He also noticed that his skin went blue very readily in the cold.

On admission to hospital he had widespread skin changes consisting of ulceration, scarring, induration, livedo reticularis and pigmentation. The active ulceration was in an arc centred on the shoulder joint posteriorly, and reaching from the acromion process back over the scapula and downwards and forwards as far as the posterior axillary line. Individual ulcers had polycyclic outlines reminiscent of gummata, but there was no other evidence of syphilis.

The scarring was a sequel to the ulceration and was found on the calves and elbows as well as the shoulders. The polycyclic outline of the ulcers was reproduced in the scars. All the scarred areas of skin were indurated, and the calves felt wooden and made biopsy difficult. The indurated skin did not pit on pressure and was not tender,

except in a few places. The injection of local anaesthetic caused great pain and subsequent biopsies were done under a general anaesthetic. Brown pigmentation of the skin bordered and outlined the white scarred areas. The indurated skin of the calves was a diffuse dark brown.

The most widespread skin change was a reticular livedo of a cyanotic colour whose pattern was exaggerated by cooling the skin. The areas most affected were those which were ulcerated, but the livedo could be traced over the skin of most of the trunk and limbs after exposure to cold.

Apart from his skin the patient was in good health and was apyrexial.

Full investigation revealed a raised sedimentation rate (35 mm. Westergren in March and 37 mm. in May), a creatinuria of 0·57 g. per litre, and an eosinophilia of 9% of 8000 white cells. Electrical responses of the muscles and the electrocardiogram were normal.

One biopsy from the shoulder, and another from the calf, showed the same changes. The epidermis was unaffected. In the sub-papillary layer of the corium there were foci of perivascular infiltration by lymphocytes and plasma cells. This infiltrate lessened in the deeper parts of the corium. The subcutaneous fat was intersected by bands of fibrous tissue. Vascular damage at this level was evident in the fibrous thickening of the vessel walls with distortion of their lumina ; one vessel appeared to have been recanalized after a thrombosis. Histology of calf muscle was normal.

The diagnosis of polyarteritis nodosa was suggested by Dr. H. W. Barber when the case was shown at the Royal Society of Medicine (Lyell and Whittle, 1949). At this time the patient developed some tender pea-sized, skin-coloured nodules of the forearms and forehead, upon a two-day-old one of which Dr. A. M. Barrett reported : " In and around the walls of several arterioles in the corium there is an intense inflammatory reaction in which polymorphonuclear neutrophil and eosinophil leucocytes are abundant. The histological picture is in agreement with the diagnosis of polyarteritis nodosa." Another such nodule excised under aseptic precautions was sterile on culture.

The patient went home in July 1949 and has been seen once subsequently, in September 1950. His general condition has improved. He has been working and has put on weight. The ulcerated areas have healed and there are now no nodules, though the livedo is still faintly visible.

Case 2.—A man aged 38 had suffered from muscular rheumatism in 1934. In 1940 he received 30 grams of sulphapyridine for gonorrhoea, in 1946 5 mega-units of penicillin for primary syphilis, and in 1946 had had a further unspecified quantity of penicillin for " septicaemia."

On 1 November 1952 he became ill with pains in the limbs and a sore throat. Two days later purplish livedo appeared on the abdominal skin and then on the skin of the right forearm. Nodules could be felt beneath the patches of livedo. At this time there were also raised painless pinkish-brown patches on both shins.

The temperature was normal and there were no other abnormal physical signs. Full investigation revealed a leucocytosis (15,500 c.mm.) with an otherwise normal blood and an E.S.R. of 10 mm. per hour (Wintrobe).

During the course of the next week there was occasional pyrexia to 99° F. Skiagrams of the chest, bones and muscles were normal. Biopsy of the skin did not help. The Mantoux reaction was negative down to 1 in 100.

The diagnosis of trichiniasis was made since a skin test for this disease gave an immediate positive reaction (20 minutes).

The leucocytosis continued (24,700 on 11 December and 20,000 on 7 January) as did the pains in his calves, though the arm pains had abated. Further skiagrams of the soft tissues of the leg were normal. New subcutaneous nodules continued to appear and one of them was excised and reported as polyarteritis nodosa by Dr. Henry Haber. ACTH was given in a dose ranging from 10 to 60 mg. daily and resulted in

clinical improvement, though the patient stated subsequently that he felt better when off ACTH.

He was later seen by Dr. Grant Peterkin. At that time there was a widespread livedo of the trunk and limbs similar to that of Case 1. There were many palpable subcutaneous nodules and a biopsy of one of these confirmed the diagnosis of polyarteritis nodosa.

The livedo and nodules continue and the limb pains persist and require 6–8 codeine tablets daily for their relief, but he feels well in himself and there has never been any evidence of a visceral spread of the disease. No ACTH has been given since May 1953.

Case 3.—A man aged 56, a core-maker by trade, had suffered from attacks of a dull pain in the left loin for about thirty years. These would last a few days every two to three months. He also had a perforated duodenal ulcer repaired in 1941 and since then had recurrent attacks of epigastric pain which was relieved by alkalis.

In October 1952 he was admitted to hospital under the care of Mr. Clifford Jones with a severe pain in the left loin similar to his previous attacks. Investigation revealed pyonephrosis as the cause of his symptoms. *Staph. pyogenes* (penicillin sensitive) was cultured from urine and blood and he responded rapidly to treatment with penicillin. He left hospital in January. Two weeks later he was readmitted (25.i.53) complaining of abdominal pain, vomiting and diarrhoea of three days' duration. The pain was relieved by a milk drip, but one week after admission the pain changed its character, becoming colicky, and he began to pass frequent loose foul stools containing frank blood.

Barium meal and enema and bacteriological examination of the faeces failed to reveal a cause of his symptoms. Two weeks after admission a red swelling appeared on the right ankle and wrist and on the following day he developed a purpuric eruption on the arms, legs and lumbar region. The abdominal pain and diarrhoea continued and the abdomen became distended.

Six weeks later (March 1953) the finger, wrist and ankle joints became swollen and painful. The swelling of the right hand and forearm and the purpuric rash persisted. He remained very ill and lost one stone in weight. In May 1953 he began to pass bright blood in his urine. He was therefore transferred for further investigation under Professor Wayne.

Investigations revealed anaemia (Hb 57%), polymorphonuclear leucocytosis (total W.B.C. 15,000), and a prolonged prothrombin time due to Factor VII deficiency. The urine contained pus, red blood cells and a penicillin sensitive *Staph. pyogenes*. The blood urea was 139 mg.%.

The anaemia responded to treatment with Fersolate, the urinary infection to treatment with penicillin and the blood urea returned to normal. The patient returned home at the end of May but two weeks later the purpuric eruption recurred. He developed oedema of the ankles and pain in the left loin followed by haematuria. He was readmitted to hospital.

One week later he developed swelling and tenderness around the flexor aspect of the left wrist which lasted two days.

His urinary infection was controlled with chloromycetin, and a four-day course of ACTH (25 mg. daily by intravenous drip) improved his general condition greatly. On 18 July a nodule appeared on the right forearm and was excised. Dr. Hermitte reported as follows: "The specimen shows bundles of voluntary muscle from the forearm some of which have undergone necrosis. In one area associated with small arteries there is an extensive acute inflammatory exudate consisting of fibrin and polymorphs with a number of eosinophils. Haemorrhage has occurred into adjacent connective tissue. The appearances indicate polyarteritis nodosa." Five days later (23.vii.53) the patient had another attack of colic and diarrhoea. Treatment with ACTH (60 mg. daily) was recommenced and the pain rapidly relieved.

The purpura faded gradually, no fresh nodules appeared and the diarrhoea ceased.

Four weeks later cortisone 25 mg. daily was substituted for the ACTH and the patient returned home on 20.viii.53.

Soon after discharge he had another relapse which was controlled with ACTH. He remains well, giving his own injections of ACTH gel, 10 mg. daily.

## Comment.

In each case biopsy of a subcutaneous nodule has revealed polyarteritis nodosa but the skin changes have differed. In Case 1 there was ulceration, scarring, livedo reticularis, induration and pigmentation, in Case 2 livedo reticularis and in Case 3 transient purpura and oedema. Apart from livedo reticularis, Cases 1 and 2 have in common muscle pains and absence of visceral involvement. In Cases 1 and 2 the skin manifestations are the disease. Compare this with the transient oedema and purpura of Case 3 whose disease is visceral and whose skin is involved incidentally.

We think that there are two forms of polyarteritis nodosa affecting the skin. One is a *cutaneous* form and is represented by our first two cases. The skin, the main organ involved, is mottled by livedo reticularis and in bad cases is ulcerated, scarred and indurated. The only suspicion of visceral involvement is the complaint of muscle pains. Case 3 represents the *visceral* form. The disease is in the internal organs, and the skin, if involved at all, suffers transient damage evidenced by purpura or other exanthemata, ecchymosis or gangrene. On account of its distribution within the body the visceral form is necessarily more serious and is often fatal whereas the cutaneous form rarely causes death.

## Summary.

The skin is involved in about a quarter of cases of polyarteritis nodosa and the possible manifestations are subcutaneous nodules, ecchymoses, gangrene, acute exanthemata especially purpura, oedema, livedo reticularis and ulceration.

We have presented three cases each confirmed by examination of an excised subcutaneous nodule and conclude that there are two forms of polyarteritis nodosa. The first is *cutaneous* and is characterized by livedo reticularis, ulceration, muscle pains and oedema, and the second *visceral* in which the skin is involved inconstantly and incidentally during the course of the disease by exanthemata, ecchymoses or gangrene. Subcutaneous nodules are common to both forms.

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## References.

Adamson, H. G. (1916) *Brit. J. Derm.*, **28**, 281.
Alkiewicz, J. (1933) *Arch. Derm. Syph., Wien*, **168**, 522.
Barber, H. W. (1949) *Proc. Roy. Soc. Med.* (*Sect. Derm.*), **42**, 945.

Barnard, W. G., and Burbury, W. M. (1934) *J. Path. Bact.*, **39**, 285.
Beattie, J. M., and Douglas, M. (1912–13) *Ibid.*, **17**, 195.
Bennett, G. A., and Levine, S. A. ((1929) *Amer. J. med. Sci.*, **177**, 853.
Bernstein, A. (1935) *Amer. J. med. Sci.*, **190**, 317.
Boyd, L. J., and Nussbaum, C. (1936) *Med. Clin. N. Amer.*, **20**, 973.
Carol, W. L. L., and Prakken, J. R. (1937) *Acta derm.-venereol., Stockh.*, **18**, 102.
Carr, J. G. (1930) *Med. Clin. N. Amer.*, **13**, 1121.
Cohen, M. B., Kline, B. S., and Young, A. M. (1936) *J. Amer. med. Ass.*, **107**, 1555.
Crosti, A. (1935) *G. ital. Derm. Sif.*, **76**, 15.
Debré, R., Leroux, R., Lelong, M., and Mlle. Gauthier-Villars (1928) *Arch. méd. Enf.*, **31**, 325.
Dietrich, K. (1933) *Z. KreislForsch.*, **25**, 305.
Dowling, G. B. (1943) *Proc. Roy. Soc. Med.* (*Sect. Derm.*), **36**, 287.
Dunbar, J. C. (1936) *Bull. Md. Sch. Med.*, **20**, 138.
Ehrmann, S. (1907) *Proc. 6th int. Derm. Congr.* (*N.Y.*), p. 763.
Freund, F. (1926) *Arch. Derm. Syph., Wien*, **152**, 158.
Frommel, E. (1926) *Ann. Méd.*, **19**, 42.
Goldschlag, F., and Chwalibogowski, A. von (1935) *Arch. Derm. Syph., Wien*, **171**, 622.
Grant, R. T. (1940) *Clin. Sci.*, **4**, 245.
Guequierre, J. P., and Greenbaum, S. S. (1942) *Arch. Derm. Syph., Chicago*, **46**, 566.
Hutinel, J., Coste, F., and Arnaudet, A. (1930) *Arch. Méd. Enf.*, **33**, 355.
Keil, H. (1938) *Medicine, Baltimore*, **17**, 261.
Ketron, L. W., and Bernstein, J. C. (1939) *Arch. Derm. Syph., Chicago*, **40**, 929.
Kimberly, L. W. (1946) *Ibid.*, **54**, 233.
Klotz, O. (1917) *J. med. Res.*, **37**, 1.
Krahulik, L., Rosenthal, M., and Loughlin, E. H. (1935) *Amer. J. med. Sci.*, **190**, 308.
Kussmaul, A., and Maier, R. (1866) *Dtsch. Arch. klin. Med.*, **1**, 484.
Lamb, A. R. (1914) *Arch. intern. Med.*, **14**, 481.
Lewis, T. (1927) *The Blood Vessels of the Human Skin and Their Responses.* London : Shaw & Sons, Ltd.
Lindberg, K. (1931) *Acta med. scand.*, **76**, 183.
——— (1932) *Ibid.*, **77**, 455.
Löhe, H., and Rosenfeld, H. (1931) *Derm. Z.*, **61**, 299.
Lyell, A., and Whittle, C. H. (1949) *Proc. Roy. Soc. Med.* (*Sect. Derm.*), **42**, 946.
Manges, M., and Baehr, G. (1921) *Amer. J. med. Sci.*, **162**, 162.
Matras, A. (1938) *Wien. klin. Wschr.*, **51**, 991.
Melczer, N., and Venkei, T. (1947) *Dermatologica, Basel*, **94**, 214.
Miescher, G. (1946) *Ibid.*, **92**, 255.
Miller, H. G. (1949) *Proc. Roy. Soc. Med.* (*Sect. Med.*), **42**, 497.
——— and Daley, R. (1946) *Quart. J. Med., N.S.* **15**, 255.
Motley, L. (1936) *J. Amer. med. Ass.*, **106**, 898.
Oliver, E. A. (1948) *Arch. Derm. Syph., Chicago*, **58**, 530.
Ormsby, O. S. (1948) *Ibid.*, **58**, 530.
Slinger, W. N., and Starck, V. (1951) *Ibid.*, **63**, 461.
Spiegel, R. (1936) *Arch. intern. Med.*, **58**, 993.
Taylor, A. W., and Jacoby, N. M. (1949) *Lancet*, ii, 792.
Unna, P. G. (1894) *International Atlas of Rare Skin Diseases.* Edited by P. G. Unna, M. Morris, H. Leloir, and L. A. Duhring. Hamburg and Leipzig : Leopold Voss. London : H. K. Lewis. Heft 10, XXXI, 2.
Vining, C. W. (1938) *Arch. Dis. Childh.*, **13**, 31.

# DEGENERATIVE CHANGES IN THE SKIN WITH SPECIAL REFERENCE TO JUTE-WORKERS

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A recent investigation into the dermatoses of jute-workers (Kinnear *et al.*, 1954), consisting of a clinical survey of 763 patients and a field survey of 3023 workers in jute establishments, has shown that (i) dermatitis is not a particular hazard of jute manufacture, (ii) oil acne is common in jute-workers but is not in itself of serious import, and that (iii) jute-workers are liable to degenerative changes of the exposed skin (face, hands and forearms), leading in a few cases to carcinoma. It is with this last finding that the present paper is concerned.

The changes we have observed in jute-workers consist of patchy pigmentation, patchy atrophy, keratoses, telangiectases, purpura and in a few cases carcinomata. The pre-malignant changes are regarded by the workers as normal senile changes, but a comparison with others including flax-workers showed that this is not so, the incidence being much higher in jute-workers than in the rest.

The pigmentation is dark, patchy and usually easily distinguished from freckles by the deeper colour, more irregular outline, size and distribution of the lesions. It bears some superficial resemblance to that found in Riehl's melanosis, of which one of us (Kinnear, 1935*a*, *b*) has recorded two cases in jute-workers. During the present investigation we looked for other examples of this condition but none was discovered.

Riehl's melanosis is characterized by reticulate pigmentation enclosing pinhead sized areas of apparently normal skin, perhaps slightly scaly. The skin of the face, the principal site, becomes markedly less involved towards the midline. Where oil is a factor in its production lesions of oil acne may be present. We can find no record of Riehl's melanosis proceeding to carcinoma of the skin; on the contrary, it tends to disappear in time. The pigmentation we refer to in jute-workers consists of more irregular patches accompanied usually by other evidence of skin degeneration including keratoses. Evidence of oil acne is usually lacking and the centre of the face is not noticeably spared.

The atrophy seen was patchy rather than general and had to be distinguished from scarring following minor injuries. Neither telangiectases nor purpura

were prominent features. Keratoses were of the "senile keratosis" type and had to be distinguished from seborrhoeic and ordinary warts. Squamous-cell carcinoma occurred in 8 jute-worker patients (including one with intraepidermal carcinoma)—5 females and 3 males –and rodent ulcer in 14—9 females and 5 males. This reversal of the normal sex distribution is probably due to the greater number of female than male jute-workers. Pre-malignant changes were seen in 6 of the 8 patients with squamous carcinomata and 8 of the 14 with rodent ulcers.

Cases of molluscum sebaceum are not included in the above numbers. This entity, also named kerato-acanthoma (Rook and Whimster, 1950) and molluscum pseudo-carcinomatosum (*Lancet*, 1953) is not an uncommon condition and statistics of skin cancer are often inaccurate by reason of its inclusion. During our clinical survey 5 cases of molluscum sebaceum were noted, 4 in jute-workers and one in a non-jute-worker. All were single lesions and all patients were over 60 years of age. In the 4 jute-workers the sites were ear, cheek, neck and forearm. In this last case it occurred on an area showing pre-malignant changes. The non-jute-worker showed a lesion on the back of the right hand on an area showing pigmentation and keratosis attributed to the effects of sunlight in an outdoor worker.

## Discussion.

The definition of normal senile changes of the skin is not easy since most of the changes appearing under this heading are influenced by extrinsic factors acting on the skin. Eller and Eller (1951) in dealing with senile atrophy state "Actinic radiation is always an important factor. The condition develops in all persons progressively during their old age, but it may develop earlier in people who have been frequently exposed to climatic and thermic stimuli". As regards the dermis, senile elastosis is in a similar position. Percival *et al.* (1949) state "with advancing years and at a much earlier age-period as a result of prolonged exposure to the elements, the skin on the uncovered areas of the body may undergo a characteristic change" (*i.e.*, senile elastosis).

We believe that purely senile changes are such as are seen on the covered parts of the body and are confined to thinning of the epidermis, partial loss of its glandular appendages, diminution in the elasticity of the dermis and loss of subcutaneous fat. Any other changes are due to the effects of extrinsic factors acting on the skin. The influence of prolonged exposure to light on different types of skin is well known; the blond blue-eyed person is much more prone to skin carcinomata than the darker skinned individual.

### *Skin Colouring and Degenerative Changes.*

During our clinical survey a note was kept of the hair and eye colouring of all patients. All could be considered as fair-skinned in that they represented the

normal mixed race of the British Isles without apparent southern strain in their ancestry, but we decided to classify them as others have done into "fair" (blue, grey, green or hazel eyes with any colour of hair) and "dark" (those with brown eyes). Fig. 1 compares our findings of the percentages of fair persons in affected and control groups with those of other workers. Those of our patients who showed malignant or pre-malignant changes are divided into "jute-workers" and "non-jute-workers". The other figures quoted show results of investigations in Europe and in the more sunny climes of California and Australia. In

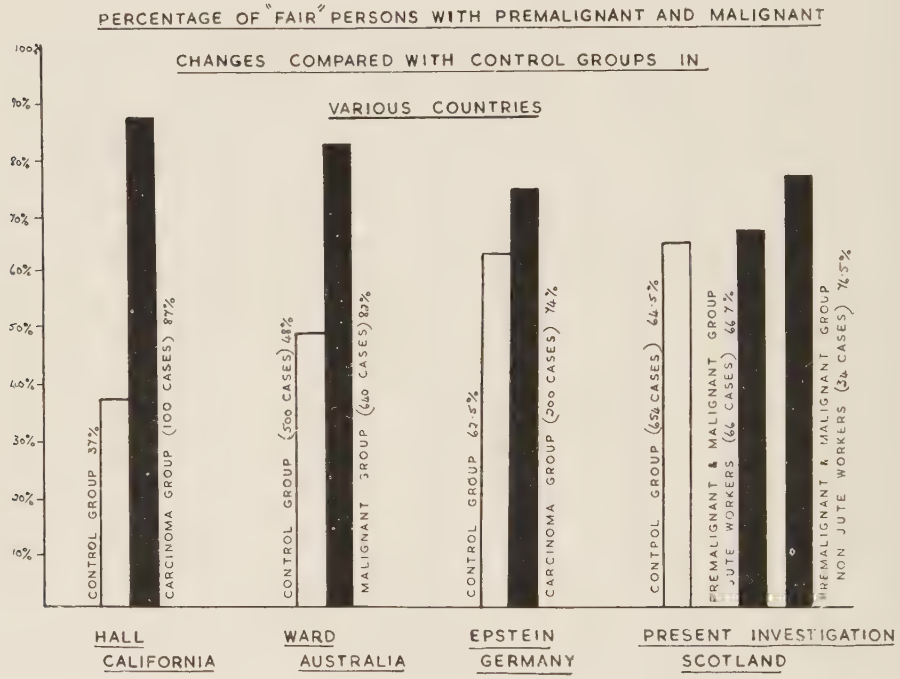


Fig. 1.

each case the control group consisted of patients who showed no malignant change.

It will be seen that our figure of 64·5% fair persons in the control group is slightly higher than Epstein's (1931) 62·5% in Germany but much higher than 48% fair in Australia (Ward, 1952) and 37% in California (Hall, 1950). We found no significant difference between the percentages of fair persons in jute-workers and in non-jute-workers. Hall's figures for 100 cases of skin cancer in California show 87% fair, Ward's for 640 cases in Australia 82% fair, and in less sunny Europe Epstein found 74% fair in 200 cases of skin cancer: our figures for malignant and pre-malignant changes in 34 non-jute-workers show 76·5% fair. Of these 34 patients 23 had been engaged in this country or abroad

in occupations with much exposure to the sun and 4 of these had in addition exposure to carcinogens such as oil or tar. Shapiro *et al.* (1953) found that in all 25 cases of squamous-cell carcinoma in coloured Bantus a definite aetiological factor other than sunlight could be established. Only one case of basal-cell carcinoma was seen. Among albino Bantus, on the other hand, the incidence was higher and of 12 cases of skin cancer (9 squamous-cell and 3 basal-cell) all tended to occur on exposed areas and most patients with squamous-cell carcinomata had other early ketatotic lesions.

The addition of another carcinogenic factor, such as mineral oil, to sunlight increases the tendency of fair persons to develop skin cancer. Schwartz *et al.* (1947) go so far as to say " it is suggested that in selecting new employees for outdoor work in the oil fields of California, marked blonds should not be chosen".

Dundee jute-workers are town dwellers and as little exposed to sunlight as any, and of 66 cases showing pre-malignant or malignant changes 66·7% were fair, a figure little higher than that for the control group. This suggests that sunlight has little to do with the occurrence of such changes in jute-workers in Dundee. Of 297 jute-worker patients 20·2% were affected against 7·4% of 458 non-jute-workers, and we believe that in jute-workers oil is the cause of these changes. Jute-workers are exposed to two types of oil—that used in lubricating the machinery and that used in the first stage of processing jute when an emulsion of oil and water is added to enable it to be spun. Although whale oil was formerly used in this " batching " process it has been entirely replaced by mineral oil, a change which has gradually taken place over many years.

### *Rodent Ulcer.*

Molesworth (1927) was the first to stress the importance of the action of sunlight, especially on a fair skin, in the production of rodent ulcer. Though our figures for rodent ulcer are too small to be used statistically it is interesting to note that the proportion of fair non-jute-workers with rodent ulcers was increased while that of fair jute-workers with rodent ulcers was the same as in the control group. This again suggests that although sunlight is a factor in producing rodent ulcers in general, it has little to do with their appearance in jute-workers.

We have seen few cases of pre-malignant changes in those employed in the other industries in which exposure to mineral oil is common, but the pre-malignant changes attributed to contact with cutting oils (Cruickshank and Squire, 1950), pitch (Jenkins, 1948), etc., appear similar to those we have described. We have encountered no lesions resembling " pitch warts " : are these not examples of molluscum sebaceum, which as we have found, may occur on altered skin ?

We have come to the conclusion that these pre-malignant changes and squamous- and basal-cell epitheliomata arise only on skin which has been acted upon by a carcinogenic agent. Probably sunlight is the commonest. Molesworth (1937) states that in Australia 99% of skin cancers are due to the effects of chronic sunburn. Previous damage to the skin, as in Shapiro's coloured Bantus, may lead to cancer, and the action of mineral oil, pitch, etc., is well known. In a highly industrialized country such as Britain, where we may encounter a variety of carcinogens in our daily life, in our roads and streets, in the atmosphere or the dust it carries, it may be difficult to identify the responsible agent, but we believe that more careful investigation will reveal a cause. We consider that attention should be paid to the type of skin in those showing changes due to carcinogens other than sunlight.

### *Oil Acne.*

One interesting observation to which we drew attention was the small number of cases in which oil acne and pre-malignant changes were found in the same person. In our field survey we found 219 (7·2%) with pre-malignant changes, 465 (15·4%) with oil acne and only 8 (0·3%) with both. Mechanics, whose work brings them especially in contact with oil, accounted for 5 of these 8 cases.

Oil acne was commoner in men than in women and its incidence decreased progressively with age while the reverse was true of pre-malignant changes. The most striking difference between the sexes was shown in the age group 60 years and over ; in this group 59·8% of women but only 9·3% of men showed pre-malignant changes. It seems that there is a change in the reaction of the skin to oil and that it gradually ceases to produce lesions of oil acne. We do not consider that different duties of men and women account for the different incidence of oil acne, but that the higher incidence in men is probably due to the larger size of their pilo-sebaceous follicles. The results of our field survey, however, do suggest that the higher incidence of pre-malignant changes in women can be related to the different type of work they perform.

### Summary.

Degenerative changes occurring in the exposed skin of jute-workers are described in detail.

The differential diagnosis is discussed and it is suggested that all pre-malignant changes in the exposed skin can be related to exposure to potential carcinogens and are not solely due to ageing of the skin.

A comparison with the results obtained by other workers shows that the incidence of degenerative changes of the exposed parts caused by sunlight is related to the type of skin. We have found that no such correlation exists in

the case of jute-workers where the carcinogen responsible is presumably mineral oil.

We are indebted to Dr. T. E. Faulkner, Lecturer in Statistics, University College, Dundee and to Dr. R. A. Main, Registrar in Dermatology, Dundee Royal Infirmary for their assistance in the preparation of this paper.

## References.

Cruickshank, C. N. D., and Squire, J. R. (1950) *Brit. J. industr. Med.*, **7**, 1.
Eller, J. J., and Eller, W. D. (1951) *Tumors of the Skin.* London : Henry Kimpton, p. 235.
Epstein, S. (1931) *Arch. Derm. Syph., Berlin*, **164**, 304.
Hall, A. F. (1950) *Arch. Derm. Syph., Chicago*, **61**, 589.
Jenkins, W. D. (1948) *Dermatoses among Gas and Tar Workers.* Bristol : John Wright & Sons Ltd.
Kinnear, J. (1935*a*) *Brit. J. Derm.*, **47**, 191.
——— (1935*b*) *Ibid.*, **47**, 374.
——— Rogers, J., Finn, O. A. and Mair, A. (1954) *Brit. J. ind. med.* In press.
*Lancet* (1953) ii, 816. (Leading Article.)
Molesworth, E. H. (1927) *Med. J. Aust.*, i, 878.
——— (1937) *An Introduction to Dermatology.* London : J. & A. Churchill.
Percival, G. H., Hannay, P. W., and Duthie, D. A. (1949) *Brit. J. Derm.*, **61**, 269.
Rook, A., and Whimster, I. (1950) *Arch. belges Derm.*, **6**, 137.
Schwartz, L., Tulipan, L., and Peck, S. M. (1947) *Occupational Diseases of the Skin.* London : Henry Kimpton, p. 238.
Shapiro, M. P., Keen, P., Cohen, L., and Murray, J. F. (1953) *Brit. J. Cancer*, **7**, 45.
Ward, W. H. (1952) *Aust. J. Derm.*, **1**, 157.

# INFANTILE ECZEMA AND ELECTRO-ENCEPHALOGRAPHY.

## CLINICAL REPORT.

W. J. O'DONOVAN, O.B.E., K.S.G., M.D.

It is the practice of modern dermatologists to look on the family as a unit to be taken note of in the consideration of any one particular case of skin disease, and in this way affections in other members of the family have thrown light upon the human type presented for clinical examination.

For long we have known of the existence of asthma, eczema, epilepsy and migraine occurring in a patient. These manifestations may be seen at different times or in association in the same patient, or a patient may show one form himself and the others of these associated maladies may be found in the surrounding or past blood relatives.

The case I am relating shows that this complex may be discoverable by a new technique when all history is unavailable. It opens the field of cutaneous research more widely and certainly indicates that more than external treatment is fundamentally necessary when a technological investigation is carried out skilfully.

### Case Report.

A slender girl of 4 years of age was brought by her mother to my Out Patient Department on 14 July 1953 with a maternal story that she had suffered for six months with infantile eczema.

She had been an in-patient in other hospitals frequently and one of her brothers suffered from chronic asthma.

On 11.ix.53, as her condition was unchanged and rather pitiful, I admitted her to hospital as an in-patient. Her mother then said she had developed this skin affection from her when she was four months old and had recently been six months an in-patient in a nearby hospital.

The child had always been nervous and showed asthma first at the age of 18 months and up to now had had three notable attacks. She had measles when very young and her mother noted that eggs, fried foods, oats and artificial creams upset her.

In her family the eldest boy aged 15 had asthma. Another boy aged 10 had had eczema when a baby. Two other boys were quite well. The mother has suffered considerably from headaches and the paternal grandfather had asthma. The home conditions were comfortable.

On admission the resident medical officer recorded her as "a dirty little girl, quite happy." The face and all the limbs showed red and moist patches which were abraded and crusted with moist and dry scabs. The lymphatic glands in the neck, armpits and groins were visibly enlarged.

By 25.ix.53 her condition had worsened. Her temperature varied about 99° F. There was no albuminuria.

She was sent by ambulance on 26.x.53 to the Director of the Encephalography Department of the Goodmayes Hospital, Essex, and there this, to me, remarkable recording was made (Fig. 1).

Accompanying this record is a significant report from the very experienced Director, Dr. J. E. Glancy, which I reproduce with his generous permission :

> " The record generally is of high amplitude. The dominant rhythm in the parieto-occipital areas is well-formed, symmetrical, at 8 c s and blocks to visual attention. It is mixed with much theta activity. This tends to be continuous in other regions and is mainly at 5 c/s. Much of it also blocks on eye opening. During the resting record there is one brief paroxysm of generalized bilaterally synchronous and symmetrical spike and wave complexes at about 3 p s. There is another during overbreathing which otherwise causes little change. Flickering light does not provoke any abnormal activity.
>
> " An abnormal E.E.G. The spike and wave paroxysms are for practical purposes diagnostic of idiopathic epilepsy."

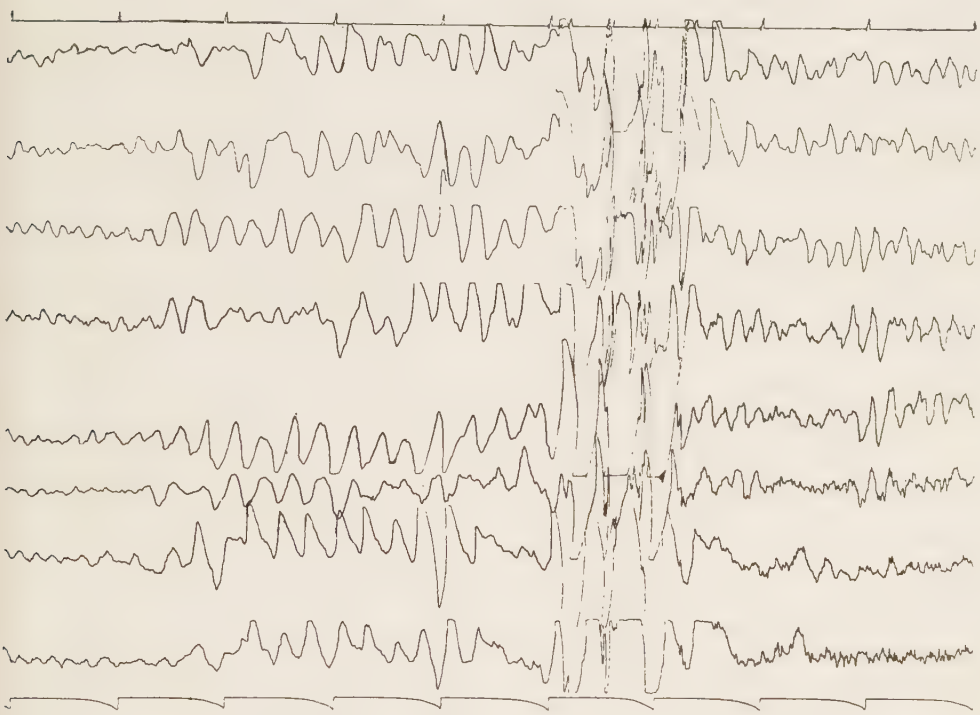

Fig. 1.

On receipt of this report the patient was given phenobarbitone one grain *t.d.s.*, and progressed remarkably well. External treatment was confined to the application of liquid paraffin. The patient's intelligence and activity was in no way blunted by the administration of pheno-barbitone.

I have not seen her since she left hospital.

## Comment.

The existence of the asthma-prurigo-epilepsy-migraine complex is widely recognized.

The changes in cerebral recordings in epilepsy are well appreciated by neurologists. This case is recorded to invite others to make similar electro-encephalographic investigations in cases of infantile prurigo or infantile eczema where the family and personal history seems lacking in epilepsy ; should such recordings be found with more frequency, then it will be appreciated that the ambit of this complex is far wider than has generally been thought.

# A SMALL EPIDEMIC OF M. GYPSEUM RINGWORM IN A PLANT NURSERY.

C. HOWARD WHITTLE.

Physician to Skin Department, United Cambridge Hospitals.

Although *M. gypseum* can infect the scalp or glabrous skin, reported cases are infrequent and mostly sporadic. Lewis and Hopper (1943) remark that the fungus appears in New York only occasionally and then usually in children. Statistics are scanty. Gaethe and Grieshaber (1950) found that only 3% of cases of scalp ringworm in New Orleans in a four-year period 1946–1949 were produced by *M. gypseum*. In New York in the same period this fungus was responsible for less than 3% of those scalp ringworm cases in which the organism was identified (Hopkins *et al.*, 1953). Sharp and Wegner (1950) discovered only 8 cases in 13 years at Galveston, Texas, and the sporadic nature of the infection suggested its introduction by shipping from South America ; but in Rio de Janeiro the incidence of *M. gypseum* infection was only 9% in a survey of 281 cases of hand and foot infection (Valerio, 1950).

In Great Britain it seems to be rare. Jacqueline Walker (1950) isolated only 8 strains out of 2473 cultures (0·3%). 7 of these were from lesions of the glabrous skin. Calnan (1952) showed one case in London, but so far as I know no others have been reported in these islands in recent years.

This report concerns a small epidemic of circinate ringworm of the glabrous skin in adults working in a carnation nursery in a country district in Hertfordshire. The men worked as packers and sorters in outhouses and about the premises. The women were mostly planting, stringing, picking and sorting the plants and flowers; they worked in the glass houses with arms and legs bare. It was in September 1951 and the weather was warm.

*The patients.*—There were eight persons affected, 4 men and 4 women, out of a population of about 40 working in the nursery. There were 11 separate lesions, two patients showing more than one lesion, and the sites affected are indicated in Table I. In those patients developing more than one lesion there was an interval of two or three weeks between the appearance of the first and second or subsequent lesion. Case I developed her three lesions in the course of four weeks.

*The lesions.*—A few patients described the eruption as starting like a " heat bump" or insect bite, with stinging and itching. The earliest lesion seen by the writer was one day old on the forearm of a man and this was already

TABLE I.

| Case. | Sex. | Date of onset. | Lesion. (E. = erythema. V. = vesicles.) | Site. | Mycology. Mycelium in films. | Mycology. Culture. |
|---|---|---|---|---|---|---|
| 1 | F. | 1.ix.51 | 1 in. E.+ V.+ | L. forearm | — | — |
| | | | ¾ in. E.+ | L. arm | — | — |
| | | 24.ix.51 | ½ in. E.+ V.+ | R. thigh | + | *M. gypseum.* |
| 2 | F. | 13.ix.51 | ¾ in. E. (healing) | L. thigh | — | — |
| 3 | F. | 13.ix.51 | ½ in. E. | L. thigh | + | Sterile. |
| 4 | F. | 20.ix.51 | ½ in. E.+ V.+ | L. shoulder | + | *M. gypseum.* |
| 5 | M. | 1.ix.51 | ⅝ in. E. (healing) | R. forearm | — | — |
| | | 1.x.51 | ⅜ in. E.+ V.+ | L. forearm | + | *M. gypseum.* |
| 6 | M. | 1.ix.51 | ½ in. E. (nearly healed) | R. forearm | — | — |
| 7 | M. | 6.ix.51 | ¾ in. E. (healing) | L. arm | — | — |
| 8 | M. | 3.xi.51 | ⅜ in. E.+ V.+ | R. forearm | + | *M. gypseum.* |

showing blisters on an erythematous base. The lesions when developed were bright red discs varying from a half to one inch in diameter with a ring of coarse vesicles at the edge. The vesicles were thick walled and did not rupture easily.

*The course.*—Some lesions had already been treated by the patient with iodine painting and elastoplast covering, and a few of these healed or were healing. They appeared to reach a maximum size of about one inch in diameter and then regress. Treatment may have speeded up the resolution but they remained active for 5 weeks or longer. The epidemic ran for a period of 2 months from the appearance of the first to the last case.

## THE CONTACTS.

*Personal.*—There was ample opportunity for fairly close personal contacts. The four women worked in the same building, often near each other. The four men were also closely associated in their work. Moreover, contacts at home or away from work were likely. For instance Cases 1 and 7 lived in the same row of houses, as did Cases 2 and 5, and Cases 6 and 8. Case 2 was the daughter of Case 4, and Case 3 the daughter of Case 8.

The earliest cases, Case 1 and Case 5, produced more than one lesion—3 in the woman, 2 in the man—which might suggest that they not only infected their own skin by transference of the fungus from the primary site but also that they may have been the source of the infection for the other cases.

*Extraneous sources of infection.*—The nursery was in the country and stood in its own grounds of about 24 acres, of which 5 acres were under glass. There

were farms and agricultural land adjoining but no apparent contact between the farm workers and nursery workers.

(*a*) Animal sources, e.g., in cattle, farm stock, domestic cats and dogs, were sought but none were found.

(*b*) Artificial manures : Some of those used were sweepings from slaughter houses. They contained animal hair, dandruff and horny matter. Infected material may show fluorescence (Gonçalves, 1953) but examination under Wood's light revealed no fluorescent particles in the samples taken, and no pathogenic fungi were grown after repeated attempts on various media.

(*c*) Imported materials or articles were sought because *M. gypseum* infections appear to be commoner in South America than in this country ; no South American products could be traced. The only articles known to be foreign were the canes used for supporting the plants and the jute sacks containing the manures. The canes were probably from China or Indo-China. This clue has not been followed up.

## Comment.

There is reason to believe that *M. gypseum* is zoophilic and is only occasionally parasitic on man. But in this epidemic there may have been contact from man to man, and the abundance of mycelium in the lesions indicates an acquired ability to grow in human skin, even though the lesions were small and tended to heal spontaneously. Even anthropophilic species may produce self-limiting and self-healing lesions. (*c.f. M. Audouini* in scalp infections, as shown recently by Kligman (1952) and Whittle (1953)).

The origin of this infection is unknown. It may be lying dormant in the soil, manure or farm buildings. There is evidence that the organism is still present in this nursery.* In this connection Ajello's work is of interest (Ajello, 1953). He found there was significant relationship between the presence of *M. gypseum* in soil samples (37 out of 116 samples) and the presence of animals at the collecting site. He suggests that it is essentially a soil-inhabiting fungus and that it may play a part in breakdown of keratin débris.

## Summary.

An epidemic of *M. gypseum* ringworm of the glabrous skin occurring in 8 adults in a plant nursery is described. Positive cultures were obtained from 4 cases.

The origin and mode of spread are discussed.

* Since this epidemic there have been further sporadic cases, and in March 1954 a considerable outbreak occurred. Thirteen cases were seen, some with multiple lesions. Positive cultures of *M. gypseum* were obtained from the six most active cases. Soil samples are receiving further study.

I should like to thank Dr. Jacqueline Walker for kindly confirming the identity of the cultures.

## References.

Ajello, L. (1953) *J. invest. Derm.*, **21**, 157.
Calnan, C. D. (1952) *Brit. J. Derm.*, **64**, 343.
Gaethe, G., and Grieshaber, F. C. (1950) *New Orleans med. surg. J.*, **103**, 256.
Gonçalves, A. P. (1953) *Ann. bras. Derm. Sif.*, **28**, 15.
Hopkins, J. G., Lingamfelter, C. S., Kiesselbach, M. R., and Hamilton, O. A. (1953) *Arch. Derm. Syph., Chicago*, **67**, 479.
Kligman, A. M. (1952) *J. invest. Derm.*, **18**, 237.
Lewis, G. M., and Hopper, M. E. (1943) *An Introduction to Medical Mycology.* 2nd ed. Chicago, Illinois : The Year Book Publishers, Inc., p. 268.
Sharp, W. B., and Wegner, M. J. (1950) *Arch. Derm. Syph., Chicago*, **61**, 824.
Trice, E. R., and Shafer, C. (1951) *Arch. Derm. Syph., Chicago*, **64**, 309.
Valério, A. (1950) *Impr. méd., R. de J.*, **25**, 47.
Walker, J. (1950) *Brit. J. Derm.*, **62**, 240.
Whittle, C. H. (1953) *Lancet*, ii, 10.
Wilson, J. W., and Plunkett, O. A. (1951) *J. invest. Derm.*, **16**, 119.

# DISCOID LUPUS ERYTHEMATOSUS FOLLOWING TRAUMA.

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As Michelson (1953) recently pointed out, almost all articles concerning lupus erythematosus commence by stating that it is a disease of unknown aetiology. MacLeod, in 1924, considered its causation under the headings of the three main theories which had characterized the trend of opinion, in chronological order, to that time : (1) The local theory, (2) the tuberculous theory, and (3) the toxic or septic theory. He discounted the tuberculous basis for the disease. It was his contention that lupus erythematosus was not a pathological entity due to one specific cause, but rather a cutaneous symptom which might be called forth by a variety of causes of a toxic or septic character. He cited instances, in discussion of the local theory, in which lupus erythematosus had appeared to occur originally at the site of a local injury, but then went on to question the validity of such observations. He did state, however, that the sun's rays might possibly determine the localization of the disease in an individual sensitized to the rays by some underlying toxaemia.

Since MacLeod's report, investigations have established the non-tuberculous nature of the disease and have tended to confirm the concept of an underlying focus of infection or toxic process as the basis for lupus erythematosus. During this 30-year period, however, occasional reports of cases in which lesions developed at sites of trauma suggests that at least sometimes local trauma may play a role. In 1926, King-Smith cited authors such as Hyde, Stelwagon, Pusey, Sequeira, Schamberg and Sutton as mentioning the fact that in some cases external irritation such as sunlight, vesication with cantharides, and bites of insects had been the exciting cause. In King-Smith's series of 49 cases, 18 gave a history of some external irritation prior to the onset ; two followed sunburn, two burns by fat, one dermatitis from a hair dye, one burn from a steam-pipe, one from a hot-water bottle burn, one from the heat of operating a theatre, two from a wound, seven from mustard gas dermatitis, and one following erysipelas. He reviewed 30 cases reported elsewhere and 11 of these followed

some external trauma ; three from exposure to the quartz light, one from dermatitis due to neo-arsphenamine, four from sunburn, one from eczema of the face, one from an insect bite, and one from erysipelas. In 1930, Appel presented a seaman who developed lupus erythematosus on the face, ascribed to exposure to the elements, and on the arm at sites of trauma due to tattooing. McCarthy, in 1931, presented a case of possible lupus erythematosus in which the lesions developed at the site of a "friction burn." Boardman, in the same year, showed a patient with possible lupus erythematosus whose lesions developed in areas traumatized by the squeezing of comedones. In 1934, MacDonald reported three cases in which discoid lupus followed trauma ; in one, lesions developed on the face over the area injured by a piece of lumber, in the second it appeared on the nose secondary to a dermatitis which followed the wearing of celluloid spectacles, and in the third the disease developed at the site of trauma from the squeezing of pustules. Franck, in 1935, reported that all six of the cases of lupus erythematosus he had seen (in Switzerland) during the previous four years were of traumatic origin, three of the patients attributing the onset to an insect bite. In 1936, Stein reported two cases in which lesions developed in operation scars. He also cited other references to the effect of trauma in inducing lupus : influence of light (Cazenave, Hutchinson, Warde, Jackson, Moeller, Haxthausen), of cold and frost (Th. Veiel, Morris and Fox, Morberg, Callomen, H. Freund), trauma of various kinds such as insect bites (Morris, Little, MacLeod, Sasaki, F. Veiel), burns (Warde, Kaposi, Siemens, King-Smith), laceration by splinters, etc. (Thibierge, W. MacDonald), operation sites, (Simpson, Schoenhof). Kilpinen, in 1946, described a patient who developed lupus erythematosus secondary to a contact dermatitis from the rubber in a mask worn at work. In 1949, Madden reported on a case of discoid lupus erythematosus with characteristic lesions of the face and in the red portion of a tattoo on the arm. Bernstein, in 1951, reported a patient who developed lupus erythematosus following a laceration by broken glass. In the same year, Spencer reported a case in which the disease appeared secondary to a dermatitis produced by hydrochloric acid. Rook and Thomas, in 1952, presented a patient who, like the one reported by Madden, developed lesions of lupus erythematosus on the face and in the red portion of a tattoo on the arm. Two possible explanations were suggested ; either that development of sensitivity to cinnabar was the essential pathogenic factor in provoking the lupus erythematosus reaction, or that lupus erythematosus provoked by some unrelated and unknown factor localized at the site of a pre-existing low-grade inflammatory focus.

## Report of a Case.

A 32 year-old white man of Italian extraction was first seen on 13 February 1953 because of a painful eruption of 10 days' duration. Examination disclosed a grouped,

vesicular dermatosis in segmental arrangement on the right side of the chest. The diagnosis of zoster was made and appropriate medication prescribed. Two weeks later the vesicles were dry, the surrounding erythema was subsiding, and pain was much diminished. Five weeks after the onset of his illness he had completely recovered, superficial whitish scars occupying the sites of many of the healed vesicles.

On 2 June 1953 the patient presented himself again because of a new eruption which had developed 22 days before, nine days after sustaining a sunburn. In

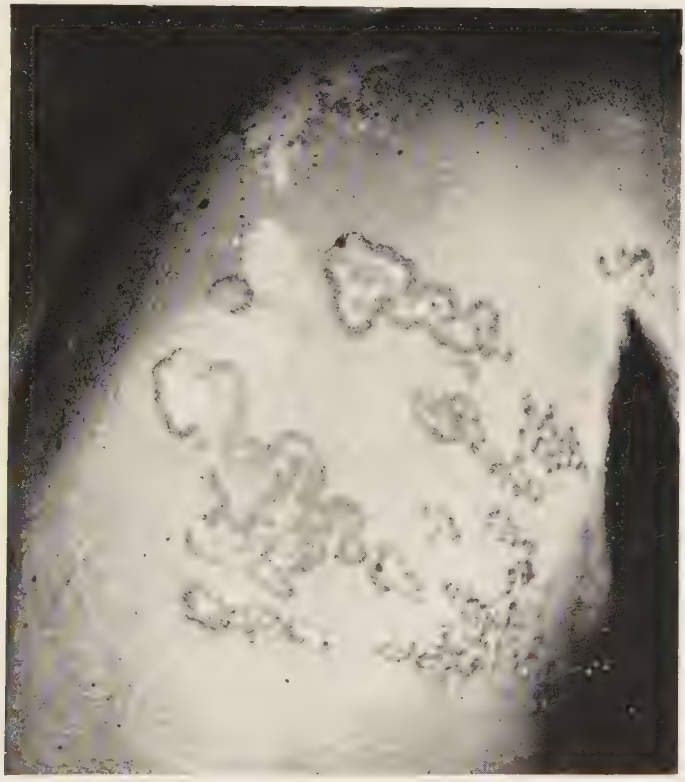

Fig. 1.

contrast to the previous dermatosis, the new one was productive of no symptoms. Examination disclosed a striking eruption which was limited to the sites of the healed zoster lesions (Fig. 1). The individual lesions were erythematous, scaly, round, with raised borders and depressed centres which showed early atrophy, telangiectasia and keratotic follicular plugging. A biopsy specimen taken from one of the lesions of the back confirmed the diagnosis of discoid lupus erythematosus. Treatment with atabrine was started, but he failed to return for re-examination.

## Comment.

The case reported is of interest because of the unusual course of events and also because it supports the concept that trauma or local skin damage may in some cases play a role in the development of discoid lupus erythematosus.

In this case there appeared to be two inciting factors, sunburn and zoster. The influence of sunlight in initiating or causing an exacerbation of the disease is well known and, with few exceptions, generally accepted. The localization of the lesions suggests that the previous herpetic eruption was responsible for some local change which made the affected sites more susceptible that the normal skin.

It appears reasonable to explain the localization of lesions in this and other cases where trauma appeared to play a role as follows: (i) An underlying predisposition to the development of lupus erythematosus as the result of a focus of infection, tissue sensitization due to earlier infections, or some other unknown cause; and (ii) a lowered resistance of those areas which had been subjected to trauma.

## Summary.

A case of chronic discoid lupus erythematosus following sunburn with the lesions limited to sites of involvement of a previous zoster is reported.

It is suggested that at least in some cases trauma of various types may play a part in the development of discoid lupus erythematosus.

## References.

Appel, B. (1930) *Arch. Derm. Syph., Chicago*, **21**, 855.
Bernstein, E. T. (1951) *Ibid.*, **63**, 261.
Boardman, W. P. (1931) *Ibid.*, **23**, 1190.
Franck, G. (1935) *Urol. cutan. Rev.*, **39**, 329.
Kilpinen, O. (1946) *Acta derm.-venereol., Stockh.*, **26**, 329.
King-Smith, D. (1926) *Arch. Derm. Syph., Chicago*, **14**, 547.
McCarthy, F. P. (1931) *Ibid.*, **23**, 1184.
MacDonald, W. J. (1934) *Urol. cutan. Rev.*, **38**, 863.
MacLeod, J. M. H. (1924) *Arch. Derm. Syph., Chicago*, **9**, 1.
Madden, J. F. (1949) *Ibid.*, **60**, 789.
Michelson, H. E. (1953) *Proc. Inst. Med. Chicago*, **19**, 335.
Spencer, G. A. (1951) *Arch. Derm. Syph., Chicago*, **64**, 215.
Stein, A. A. (1936) *Urol. cutan. Rev.*, **40**, 654.
Rook, A., and Thomas, P. J. B. (1952) *Brit. J. Derm.*, **64**, 62.

MAY 2 1954



# ROYAL SOCIETY OF MEDICINE.

## SECTION OF DERMATOLOGY.

President.—Dr. R. T. Brain.

17 December 1953.

**Acridines in the Treatment of Mycosis Fungoides.**—Dr. C. H. Whittle and Dr. J. L. Moffatt.

A man aged 68 (retired).

*History.*—First seen 22 August 1946 with seborrhoeic type eruption on neck and groins for past 5 years.

31 December 1946 : admitted to hospital. The skin of neck, extensor surface of elbows, groins and supra-natal region showed thickened, infiltrated, scaly erythema ; very itchy. Scratch marks on back and lichenification on inner sides of legs.

Blood count : Eosinophilia 800 per cu. mm.

E.S.R : 9 mm.

Biopsy : Histology supports the diagnosis.

*Course.*—He was given a course of daily intravenous injections of 25 cc. of 2% euflavine (Muller, 1937). In 3 days the lesions began to fade and the skin became deeply tinged yellow. He was ill for a few days but recovered and left hospital. The lesions faded and flattened though they had not then completely disappeared. He was apparently free of symptoms and lesions till 1953.

12 November 1953 : Histology shows pleomorphic cellular infiltration and supports the diagnosis of mycosis fungoides.

*Present condition.*—An area over the lumbar region of bluish-brown plaques slightly infiltrated, with two circular discs 2″ and 1½″ diameter on right side, of depigmented skin without atrophy.

*Comment.*—This was our first patient with mycosis fungoides in which acridine compounds appear to have promoted resolution of lesions, in this instance with 7 years' remission. Three other patients whom we showed at the November 1953 meeting of this Section were treated with another acridine compound, mepacrine by mouth, with similar resolution of lesions. Mepacrine is less toxic than euflavine, P32 or nitrogen mustard and easier to give. Spontaneous remissions in this disease are well known, but we think the remission was probably brought about by the acridine compound used.

Reference.

Muller, H. (1937), quoted by Schaeffer, J., *Behandlung der Haut und Geschl. Krankheiten.* 9th Edn.

**Discoid Lupus Erythematosus : Mepacrine Sensitivity.**—Dr. P. A. J. Smith (for Dr. W. N. Goldsmith).

A woman, aged 53 years.

The patient developed discoid lupus erythematosus of the face and occiput while having treatment in 1948 for hypochromic anaemia, dysphagia and sore mouth and

tongue of 4 years' duration (fersolate, nicotinic acid, riboflavine). For the lupus erythematosus she received numerous treatments which neither cleared all the old lesions nor prevented the formation of new ones.

May August 1948 : Myocrisin, 14 weekly injections. Total 0·295 g. After the last injection she developed a red scaly interscapular and sternal rash and erythema of the abdomen. This disappeared within a month.

October 1948–June 1949 : Bismuth, 13 weekly injections. Total 2·6 g.

December 1948–March 1950 : Bismuth, 9 weekly injections. Total 1·8 g. Local pain and swelling followed several of the injections. The face was now healed but the occiput still red and scaly.

August 1950–November 1950 : Quinine bihydrochloride 5 grains *b.d.*

January 1951 : Sore tongue rapidly improved by riboflavine and nicotinic acid.

March–May 1951 : N.A.B., 6 weekly injections of 0·45 g. Pruritic macular rash developed all over the body a few hours after the last injection, also nausea and anorexia. Quite recovered in 2 weeks.

Tongue sore again but responded in a few days to ascorbic acid. New areas of lupus erythematosus on the face and vertex.

December 1951–April 1952 : Pyridoxine, 180 mg. daily. New lesions of lupus erythematosus developed during this treatment.

March 1953 : Mepacrine, 200 mg. daily. On the third day there was generalized urticaria and swelling of the face and eyelids. Mepacrine stopped.

May 1953 : Mepacrine 200 mg. daily. Stopped after 3 days on account of nausea, abdominal pain and aching in the legs.

New lesions of lupus erythematosus developed in July and August.

17 September 1953—Mepacrine 100 mg. daily.

24 September 1953—Mepacrine 200 mg. daily. After 3 weeks of mepacrine the skin was deeply yellow and the lupus erythematosus lesions were breaking up.

After 7 weeks treatment (5 Nov.) the lupus erythematosus was not visible. At this time the *hands* became red, tense and tender. Two days later the *tongue and mouth* became sore and mepacrine was stopped after 3 days. In the following 4 weeks the tongue became more tender and ulcerated and swallowing was painful. The discomfort of the hands increased and the feet were slightly tender. There were no general symptoms.

*On examination* (12 December).—Mepacrine staining well marked. Lupus erythematosus not apparent.

*Buccal mucosa* : widespread bluish-white reticulation as in lichen planus. Triangular ulcerated area on left edge of tongue.

*Palms and flexor surfaces of fingers* : generalized hyperkeratosis and violaceous erythema with fissures in the creases. White macules 2 mm. or more in diameter form confluent areas especially over thenar and hypothenar eminences.

*Dorsa of hands and distal half of flexor aspect of forearms* : blotchy violaceous erythema, slightly infiltrated in some areas, and showing in places a whitish reticulation suggestive of Wickham's striae.

*Feet* : erythema and hyperkeratosis of soles. Scaling of heels and sides of feet.

*General physical examination* : no abnormality.

*Investigations.*—Urine : No albumin ; no cells or casts. W.B.C. : 6,200 ; Polymorphs 59%, lymphocytes 29%, monocytes 9·5%, eosinophils 1%, mast cells 1·5%. E.S.R. 16 mm. in 1 hour. R.B.C. 4,000,000. Hb. 82%. C.I. 1·03. M.C.D. 7·9

Dr. Hellier : I am interested in this sensitivity and question whether this is sensitivity in the true allergic sense. The general opinion in American literature is that the development of the rash depends to some extent on the total dose. Some people react more quickly than others, but the implication is that if a big enough dose is given most people will react. The smaller the dose the less likelihood there is of a reaction. If it is a toxic and not an allergic manifestation, one should be able to persist with treatment at a fairly safe dose. Is the lichen planus-like rash really lichen planus ? In mepacrine dermatitis one gets a complete gradation from exfoliative

dermatitis to lichen planus or a seborrhoeic dermatitis-like eruption. If the mepacrine rash is lichen planus and if lichen planus is a specific entity, it is difficult to understand this series of pictures. If on the other hand lichen planus is a skin reaction allied to other reactions, one can see how mepacrine can bring out a reaction sometimes more like seborrhoea and sometimes more like lichen planus. Lichen planus would thus be in the class of reactions comparable with eczema, psoriasis, etc.

Dr. D. S. Wilkinson : I have looked up all our cases, most of whom have been treated with 100 mg. mepacrine a day, and we had quite a high proportion of minor transient reactions as well as the case I have shown to-day, and an agranulocytosis.

Dr. J. Ferguson Smith : On the question of whether this is a true lichen planus or not and whether mepacrine and other toxic eruptions are the same I have seen one case myself and I know of one striking case from the German literature (Wirz, F., 1924, *Derm. Z.*, **41**, 269) where a single lichen planus lesion existed before any drug was given, which made it probable that the extensive eruption which developed after bismuth was true lichen planus activated by the bismuth.

Dr. W. N. Goldsmith : Lichen planus, even when *not* provoked by an ascertainable cause such as a drug, can begin with an eruption resembling pityriasis rosea. An interesting feature in our case was the variation in type of reaction and in the latent period between exhibition of the drug and beginning of symptoms on the different occasions.

The President : In my clinics we have seen very few of these toxic rashes which we could attribute to mepacrine.

**Keloids and Acromegaly.**—Dr. P. J. Hare.

Man aged 39, a decorator.

Keloids have been developing for about 10 years. There is no history of injury or disease of the skin other than acne vulgaris in adolescence. The keloids itch and cause scratching. It is uncertain how long the acromegaly has been present. It was briefly noted in 1949 when he attended hospital for a fractured toe.

*On examination.*—Across the chest and over the shoulders and back are numerous brownish-red, smooth-surfaced, firm, elevated, linear lesions about 0·5 cm. wide, and up to several centimetres in length. There are rounded and linear atrophic areas of about the same dimensions running in the same directions as the keloids, and in some instances confluent with them. There are small pitted scars and a patch of seborrhoeic dermatitis on the back. There are also small molluscа pendula on the neck and in the axillae. He shows marked general features of acromegaly.

*Investigations.*—Histology of the keloid and contiguous atrophic area : The keloid shows a spindle-shaped area in the midcutis containing plump fibroblasts and young collagen. There is metachromasia with thionin, and this largely disappears after treatment with hyaluronidase (Benger's Hyalase). There is also alkaline phosphatase activity in this area. The adjacent atrophic area shows felting of the elastica, the fibres of which run parallel to the skin surface.

Skiagrams (2 October 1953) : Show enlargement of the sella turcica and air sinuses. The hands show enlargement and thickening of the phalanges. Skiagram of the right foot (22 August 1949) : Shows narrowing of the bones of the phalanges with a fracture of the 4th proximal phalanx. The urine contains no reducing substance. Glucose tolerence test (8 October 1953) : Normal. Twenty-four hour excretion of 17-ketosteroids (10 October 1953) : 4·9 mg (? valid).

*Discussion.*—It may be a coincidence that this man has both acromegaly and keloids, but this association has been described. The atrophic areas may represent involuted keloids. The presence of hypertrophic and atrophic lesions of the skin at the same time does not rule out the possibility that the keloids may be due to an excess of somatotrophic hormone. A parallel state of affairs is seen in the bones, some of which are hypertrophic as in the hands. In the feet, there are characteristic pipe-stem phalanges and osteoporosis of the metatarsal heads

Histologically the results are similar to those obtained by Kellgren and co-workers in their study of the skin of acromegalics. No metachromatic material was detected, except in the keloids, as has been described by Asboe Hansen.

References.

Kellgren, J. H., Ball, J., Tutton, G. K. (1952) *Q.J. Med.*, New Series XXI, **84**, 405.
Asboe Hansen, G. (1951) *Acta derm. venereol.*, **30**, 221.

Dr. R. D. Sweet : I have recently seen an almost exactly similar condition in a lady aged 50, who has acromegaly, and about a dozen spontaneous keloids, mostly on the upper part of her back.

The following cases have been published in *Proc. R. Soc. Med.*, **47**, 401 :

**Poikiloderma with Epitheliomata.**—Dr. D. S. Wilkinson.

**Werner's Syndrome.**—Dr. D. S. Wilkinson.

**Familial Primary Self-healing Squamous Epitheliomata of the Skin** (Ferguson Smith).—Dr. J. S. Pegum.

The following cases also were shown :

**Lamellar Desquamation of the Newborn** (**Foetal Ichthyosis**).—Dr. J. E. M. Wigley.

**Kerato-Acanthoma of the Right Ear.**—Dr. J. L. Moffatt and Dr. C. H. Whittle

**Dermatitis Artefacta.**—Dr. R. D. Sweet.

**Lupus Erythematosus with Unusual Buccal Lesion : Loeffler's Syndrome after Mepacrine.**—Dr. D. S. Wilkinson.

**Porokeratosis of Mibelli.**—Dr. R. G. Howell (For Dr. E. W. Prosser Thomas).

**Argyria.**—Dr. R. G. Howell (For Dr. G. B. Dowling).

**Scleredema Adultorum of Buschke.**—Dr. D. L. Rees.

**Scleredema Adultorum of Buschke.**—Dr. K. D. Crow.

**Late Lues.**—Dr. N. A. Thorne.

# BOOK REVIEWS

## The Year Book 1953–1954.*

The present edition of this very successful publication incorporates abstracts of articles taken from the journals over the period December 1952 to November 1953. As the authors point out in their introduction, the increasing volume of medical literature renders their task of selection a more difficult one, but they have achieved it with conspicuous success.

The authors' own chapter this year is devoted to an assessment of advances which have occurred in treatment since the war. In thirty pages they assess the value of sulphonamides, antibiotics, quinolines, antihistamines and various other new treatments that have come to occupy some place in dermatology in recent years. Most dermatologists would agree that many of the new ointment vehicles such as carbowax have proved disappointing, but that the oil in water emulsions, and bases of the class of Ung. Aquosum (B.P.) have established their value.

It is interesting that the authors mention grenz rays at some length and advocate their use on a wider scale than hitherto. Other subjects which are discussed are the treatment of lupus erythematosus with mepacrine and chloroquine, and the stimulation and inhibition of pigmentation with xanthotoxin and monobenzyl-hydroquinone respectively.

The remaining sections of the book, comprising several hundred abstracts from the dermatological literature of the year, continue the high standard we have come to expect of this annual publication. F. R. B.

## Mycology.†

This is really the mycological section of the *Handbook of Tropical Dermatology and Medical Mycology*, Vol. II, which has already been reviewed (*Brit. J. Derm.* **66**, 256.) It is now appearing in a bright new cover with the addition of chapters on the development of medical mycology and on the doubtful fungous conditions verticilliosis and scopulariopsidosis. There is also a useful series of photographs of culture plates, many in colour. H. T. H. W.

## "You and Your Skin."‡

"Physicians may wish to keep four or five copies . . . in circulation among patients under their care."

This remark on the cover seems to state the object of the book, and the author is at his best when he is answering questions which are usually asked by patients

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* *The Year Book of Dermatology and Syphilology*, 1953–54 Series. Edited by Marion B. Sulzberger and Rudolf L. Baer. The Year Book Publishers, Inc., Chicago, Illinois. Pp. 456. Illus. 54. Price $6.00

† *Medical Mycology*. By R. D. G. Ph. Simons. Amsterdam : Elsevier Publishing Co. Distributors Cleaver-Hume Press, London, 1954. Pp. 446. 257 figs. Price 55*s*.

‡ *You and Your Skin*. By Norman R. Goldsmith. Springfield, Illinois : Charles C. Thomas, 1954. Oxford : Blackwell Scientific Publications. Pp. 148. Price 27*s*. 6*d*.

suffering from the common skin diseases. His advice in the chapters on acne, allergy, moles and other naevi, and the hair, is in accordance with accepted dermatological practice, and certain patients would find them readable and informative, though perhaps somewhat racy.

There is a good chapter on syphilis (" When boy meets girl and the spirochaetes begin to dance, what happens ? "), but it is in the form of a popular public health lecture ; there would seem to be little reason to recommend it to a patient who has already sought medical care.

Much of the rest of the book is composed of accounts of the common skin diseases, often very brief. These sometimes resemble two-minute answers in a junior examination paper for nurses : most of the information would be better given by a good physician, and the pitfalls of dermatological diagnosis and treatment make it highly undesirable for patients to try to use such a text as a substitute for medical attention. Some of the remarks could be terrifying to a patient who knew his diagnosis : " In chronic cases " of scleroderma " the face may become so involved that a hawk-like mask results " ; or " The chronic form " of lupus erythematosus " may become the acute which can be fatal".

Though one might recommend the book to the occasional patient whose demand for explanations outran one's time or patience, its value to the English dermatologist would not seem very great ; its disadvantages as a text for the layman would be lessened by drastic cutting. R. E. B.

# CURRENT LITERATURE

**TREATMENT BEFORE DIAGNOSIS IN VENEREOLOGY.** R. R. Wilcox. (1954) *Brit. J. vener. Dis.*, **30**, 7.

Arguments are presented by the author for this " cart before the horse " method of approach but much of the evidence has a distinctly sandy basis, is far from convincing, and incurs the risk of a loss of prestige to the venereologist. W. G. A.

**FOR AND AGAINST TREATMENT BEFORE DIAGNOSIS.** A. J. King. (1954) *Brit. J. vener. Dis.*, **30**, 13.

The author is against treatment before diagnosis as, purely on the grounds of common sense, it is better to know where one is going before one goes. The case is presented concisely and lucidly and, in the discussion which followed, the vast majority present were in complete agreement with Mr. King. W. G. A.

**APLASTIC ANAEMIA AND SEVERE HEPATIC ZONAL NECROSIS AFTER ANTI-SYPHILITIC TREATMENT.** A. W. B. Edmunds. (1954) *Brit. J. vener. Dis.*, **30**, 45.

The case of a male aged 53 is reported in which organic arsenical therapy was followed by both aplastic anaemia and a severe hepatitis, probably due to the virus of homologous serum jaundice. W. G. A.

**MULTIPLE PERIPHERAL NEURITIS.** E. M. C. Dunlop. (1954) *Brit. J. vener. Dis.*, **30**, 44.

The case of an Indian seaman aged about 35 years is briefly reported because of the interesting diagnostic problem it presented. The patient was referred to hospital because of ulcers on his hand thought to be due to syphilis. Trophic ulceration due to multiple peripheral neuritis caused by leprosy was diagnosed clinically and subsequently confirmed. W. G. A.

**SYPHILIS IN PREGNANCY.** Chaim Berlin and Clara Meyrovitz. (1954) *Brit. J. vener. Dis.*, **30**, 28.

During the eight years 1944–52, routine serological blood examination of 37,540 pregnant women revealed 153 positive or doubtful reactions. Types of treatment and results are discussed and the authors conclude that penicillin given to an expectant mother has superior therapeutic effects and offers greater safety for the child. W. G. A.

**ACQUIRED SYPHILIS IN CHILDREN.** Elisabeth Rees. (1954) *Brit. J. vener. Dis.*, **30**, 19.

Six cases of acquired syphilis in children and 1 possible reinfection are reported. These occurred in a small female clinic during the 7 years 1946–1953, during which 10 new cases of congenital syphilis in children of 14 years and under and 45 cases of primary and secondary syphilis in adults were diagnosed. These figures are too small to be of statistical importance, but they tend to support the belief expressed by several writers that the possibility of the asexual transmission of syphilis should receive greater attention. The author suggests that in every instance routine examination and follow-up of the family contacts of patients with early infectious syphilis should be carried out. W. G. A.

**THIRD-GENERATION SYPHILIS AND BILATERAL INTERSTITIAL KERATITIS OCCURRING IN ALL THE SURVIVING MEMBERS OF TWO GENERATIONS.** F. S. Bonugli. (1954) *Brit. J. vener. Dis.*, **30**, 24.

A detailed description is given of two cases of third-generation syphilis occurring in the same family. The coincidental findings of interstitial keratitis in all the surviving members of two generations of this family is also noted. Finally, the typical slow insidious evolution of luetic manifestations extending over a quarter of a century in a hitherto " unmanaged " family group is described. W. G. A.

**SYSTEMIC DONOVANIASIS.** R. J. Rajam, P. N. Ranhiah and V. C. Angüli. (1954) *Brit. J. vener. Dis.*, **30**, 73.

A brief view of systemic Donovaniasis is essayed and a case is presented in which the initial involvement of the cervix uteri was subsequently followed by involvement of the bladder resulting in vesico-vaginal fistula, and metastases in the bones and liver. It is claimed that this is the first instance in which the organisms of the disease were demonstrated in the liver cells of a living patient. W. G. A.

**DISSECTING ANEURYSM OF THE AORTA. DIFFERENTIAL DIAGNOSIS FROM SYPHILIS.** G. O. Horne. (1954) *Brit. J. vener. Dis.*, **30**, 88.

The author states that dissecting aneurysm of the aorta has frequently been mistaken for syphilitic aortic disease. After a description of the chief clinical features of dissecting aneurysm and a review of the beliefs held on its pathogenesis and mechanism, the differential diagnosis from syphilis, both ante-mortem and post-mortem, is discussed. Three case histories (one with post-mortem report) illustrate the difficulties encountered in differential diagnosis and the author mentions that, if the current decline in the incidence of syphilis continues, there is likely to be a relative increase in the incidence of dissecting aneurysm. W. G. A.

**THE TREATMENT OF RINGWORM OF THE SCALP.** A. R. Cofano. (1953) *Dermatologia*, **4**, 110.

A full and carefully worked-out series of cases treated with fungicidal ointment alone—inert substances alone—both these following manual epilation, and x-ray epilation alone. They find the last method by far the most satisfactory for the highest percentage of cures. They did not find a significantly higher proportion of cures in those treated with fungicidal as opposed to inert ointments. There was a preponderance of infection on the right side as opposed to the left side of the scalp, probably due to rubbing or scratching. D. S. W.

**PURPURA ANNULARIS TELANGECTOIDES: THREE FAMILIAL CASES.** G. Borelli. (1953) *Arch. ital. Derm. Sif.*, **25**, 259.

An interesting group of patients in one family. A full description of the appearance and investigation made in each case. The predilection for the thighs and buttocks is shown in the photographs. All the investigations were negative except for some indication of an increased capillary fragility. One patient was treated with ACTH with some improvement, though this was regarded as temporary. D. S. W.

**LOCAL ACTION OF CORTISONE ON CUTANEOUS REACTIVITY.** L. Rasponi. (1953) *Arch. ital. Derm. Sif.*, **25**, 275.

Description of the modifications brought about by intravenous cortisone on various forms of cutaneous reactivity, such as that to tuberculin and to ultra-violet light.

The reduction in reactivity is brought about by the general rather than the local action and naturally after a certain delay. D. S. W.

**CHROMOBLASTOMYCOSIS (DERMATITIS VERRUCOSA): REPORT OF A CASE.** Brian H. R. Hill, L. G. Cook and C. Saxby. (1953) *Austral. J. Derm.*, **2**, 80.

The patient concerned is thought to be the first case of the kind to be reported from New Zealand. R. E. B.

**X-RAY THERAPY OF PLANTAR WARTS: A REVIEW AT SEVEN YEARS OF TWO HUNDRED AND TWENTY-ONE CASES.** Adrian Johnson. (1953) *Austral. J. Derm.*, **2**, 83.

221 cases of plantar warts were treated by irradiation in 1946; 200 were traced and reviewed in 1948; 178 were traced and seen in 1953.

The cure-rate of those over 25 years old at the time of treatment was about half that of those under 25 years.

Reasons are given for concluding that some of this difference may be the result of faulty diagnosis resulting in the irradiation of callosities. R. E. B.

**SOME CHARACTERISTICS OF THE CONTACT-TYPE ALLERGIC ECZEMATOUS PROCESS IN MAN. A DISCUSSION OF ITS COMPARATIVE IMMUNOLOGY AND MORPHOLOGY.** Marion B. Sulzberger and Victor H. Witten. (1953) *Austral. J. Derm.*, 2, 57.

This paper is a summary of a vast amount of work on its subject, and does not lend itself to further condensation. It has 61 references to the literature. R. E. B.

**GENERALIZED EXFOLIATIVE DERMATITIS AND LIVER FUNCTION.** F. Bauer. (1953) *Austral. J. Derm.*, 2, 69.

The association of generalized exfoliative dermatitis with depressed liver function is frequently mentioned in the literature, but it is one which is not clearly defined, probably owing to its inconstant occurrence and to the large number of tests for liver function.

This paper is mainly an attempt to decide whether any physiological or anatomical deviation from normal is the cause or the result of the dermatosis. Investigation of ten cases showing widespread exfoliation from various causes revealed a depression of the serum albumen levels and of the hippuric acid excretion as compared with ten controls. The scale of normal values accepted in the laboratory concerned is not mentioned.

Reasons are given for concluding that the skin lesions were responsible for the liver disturbance. Two livers were studied histologically, one after biopsy and one at necropsy. R. E. B.

**THE DETECTION OF CANDIDA ALBICANS.** R. Christie and Mary M. Morton. (1953) *Austral. J. Derm.*, 2, 87.

1. A method of using guinea-pigs and mice to detect pathogenicity in strains of *C. albicans* is described.

2. Agglutination of *C. albicans* by the slide method has been shown to be reliable.

3. Seventy-eight strains of yeast-like organisms have been submitted to a number of tests to decide upon a method of identifying *C. albicans*. The tests were fermentation reactions, formation of hyphae and chlamydospores in cornmeal extract, animal pathogenicity tests, slide agglutination with an absorbed anti-serum and colony appearance on eosin-methylene blue agar. No single test was completely accurate.

4. Eosin-methylene blue agar is a valuable medium for isolating and detecting *C. albicans*.

5. Slide agglutination gives the smallest number of errors where a reasonably rapid decision has to be made.

The above is the summary given by the authors who present their facts clearly, and to whose paper it is necessary to refer for details of their techniques. R. E. B.

**BOWEN'S DISEASE OF BUCCAL MUCOSA.** H. Reich. (1954) *Arch. Derm. Syph. Berl.*, **197**, 145.

Only 7 cases could be found in the literature. The lesion, papillomatous and hyperkeratotic, affected upper and lower lip on the right side and extended over the right buccal mucosa, hard palate and upper gum. Lymph node enlargement was present. From the photograph one would have suspected squamous epithelioma, but the histology was that of papillomatous, vegetating Bowen's disease. Radiotherapy was ineffective. The patient, a 59-year-old woman, died. Bowen's disease of the buccal mucosa is a dangerous condition. M. F.

**A CONCEPT AND THERAPY OF PRURITUS ANI IDIOPATHICUS.** J. Ferreira Marques. (1953) *Arch. Derm. Syph. Berl.*, **197**, 85.

The trouble is regarded as a faulty secretion of sebaceous and apocrine glands which irritates the nerve endings. Massive vitamin A therapy provides the answer. M. F.

**EXPERIMENTAL AND CLINICAL EXPERIENCE OF ISONIACID THERAPY OF SKIN TUBERCULOSIS.** J. Meyer-Rohn and K. H. Schulz. (1954) *Arch. Derm. Syph. Berl.*, **197**, 160.

A modified Dubos medium was used to culture tubercle bacilli. Good clinical results were obtained as in other published series; but living, virulent tubercle bacilli were grown from excised skin of lesions clinically healed and showing, on section, no evidence of tuberculoid formation. M. F.

**CONCERNING SO-CALLED THROMBOPHLEBITIS MIGRANS.** M. Ruiter. (1953) *Arch. Derm. Syph. Berl.*, **197**, 22.

As a result of detailed histological examination an allergic aetiology is postulated. M. F.

**PULMONARY TUBEROUS SCLEROSIS.** John Dawson. (1954) *Quart. J. Med.* (N.S.), **23**, 113.

A full and detailed review of the history and diverse features of the syndrome tuberous sclerosis. The incidence is estimated at one in 150,000. Pulmonary manifestations are uncommonly found in association with mental signs. One of seven cases of adenoma sebaceum had pulmonary changes. The author adds four new cases of pulmonary tuberous sclerosis to the nine already recorded. The morbid lung changes include a profuse overgrowth of connective tissue, including smooth muscle and blood vessels, leading to the formation of numerous small cysts. There is a photograph of the moulage of adenoma sebaceum made for Dr. Addison at Guy's Hospital in 1851. There are also excellent photographs of lumbar peau chagrine and subungual fibromas.

J. M. B.

**INCONTINENTIA PIGMENTI.** A. Fronceschetti and W. Jadassohn. (1954) *Dermatologica*, **108**, 1.

The authors hold that incontinentia pigmenti can be divided into two groups. The first, which occurs almost exclusively in females, is hereditary, though its genetic type is obscure. It is characterized by splash-like pigmentation, ocular troubles, variable and often of malformative type, absence or malformation of teeth, and alopecia of the type of pseudo-pelade. The second group includes both sexes, and shows hereditary transmission as a dominant. Its features are a reticular pigmentation, discomfort from heat owing to poor activity of sweat-glands, dental abnormalities—bad teeth, with yellowish spots—and keratosis plantaris and palmaris. J. F. S.

**THE AETIOLOGY OF "KNUCKLE-PADS."** A. Veltman. (1954) *Dermatologica*, **108**, 29.

"Knuckle-pads" were found in a man, his son and his daughter, without any suggestion of an occupational or other local cause, or indeed any cause other than inheritance. J. F. S.

**SELF-INFLICTED LESIONS.** C. H. Beek. (1953) *Dermatologica*, **107**, 114.

Beek analyses 34 cases of self-inflicted lesions of the skin seen in the past ten years. He divides them into two main groups, the purposeful, deliberate or compensation cases, and the psychoneurotic, in which the motive was subconscious, particularly a craving for sympathy. Four of the 7 in the first group were males, but all the 27 in the second group were females, 18 being 21 years of age or younger, and 16 in their teens. Two of the older patients, aged 30 and 42 respectively, required psychiatric treatment, and two of the girls had true epilepsy. In the remainder, simple explanatory talks were curative. J. F. S.

**THE RELATIONSHIP OF MORBUS RECKLINGHAUSEN AND EHLERS-DANLOS SYNDROME.** F. Okmea and M. Depaoli. (1954) *Dermatologica*, **108**, 165.

Many divergent opinions have been expressed about a possible connection between von Recklinghausen's disease and Ehlers-Danlos syndrome, and the bibliography attached to the present article is astonishingly copious. An account is given of a lad of 16 who showed fairly extensive lesions of von Recklinghausen's disease—neurofibromata and café-au-lait patches—and, in his right arm only, cutis hyperelastica and overflexibility of the joints. The elastic fibres in the neighbourhood of the neurinomata showed hyperplasia and elastorrhexis, which are considered to be characteristic of Ehlers-Danlos syndrome. The authors also noted in the nerve fibres degenerative and proliferative changes which are regarded as stages of development of the one pathological process, and not as separate processes. They conclude that in the case observed it was not an accidental simultaneous occurrence of two different diseases, but parallel results of one primary disorder of embryonal development, which affected both ectoderm and mesoderm, and thus led to changes which belong to both Morbus Recklinghausen and Ehlers-Danlos syndrome.

J. F. S.

**ACTH AND CORTISONE IN THE TREATMENT OF KELOIDS.** F. Ronchese and A. B. Kern. (1954) *New Engl. J. Med.*, **250**, 238.

Five patients with keloids were treated with an ointment containing cortisone during the post-operative period, and 3 also received ACTH or cortisone, or both, intramuscularly or by mouth.

Although the excised keloids returned in all patients, the result in one was excellent. Ointment alone appeared to be without effect.

After reviewing the literature and considering their own cases, the authors conclude that ACTH and cortisone administered systemically may be of value when combined with surgery, but should only be tried when surgery, followed by radiation, has failed. A. D. P.

**CUTANEOUS ANERGY AND HODGKIN'S DISEASE.** H. W. Schier. (1954) *New Engl. J. Med.*, **250**, 353.

Cutaneous anergy was found in 33 cases of Hodgkin's disease to the antigens of mumps virus, candida albicans, trichophyton gypseum and purified protein derivative of tuberculin.

The reaction to histamine was normal. Attenuated reactions were noted in chronic myelogenous and lymphatic leukaemia, lymphosarcoma and reticulum-celled sarcoma.

It is suggested that patients with Hodgkin's disease fail to produce demonstrable antibodies in normal quantities. A. D. P.

**GENERALIZED PURPURIC ECZEMATOID.** P. Temime. (1954) *Ann. Derm. Syph., Paris*, **81**, 152.

A description of a man aged 52 who had a progressive widespread slightly scaling purpuric eruption. Histologically the picture was essentially eczematous. The diagnosis and aetiology are discussed. Some improvement occurred with vitamin P. F. F. H.

**THE RADICAL CURE OF FRECKLES WITH A PENCIL OF CARBON DIOXIDE SNOW (100 CASES).** Matarasso. (1954) *Ann. Derm. Syph., Paris*, **81**, 166.

The technique, advantages and disadvantages of this form of treatment are discussed. F. F. H.

**RETICULAR PIGMENTARY DERMATOSES OF THE FLEXURES.** R. Degos and B. Ossipowski. (1954) *Ann. Derm. Syph., Paris*, **81**, 147.

A woman aged 32 has presented since 1940 a slowly increasing reticular pigmentation of the major folds and under the breasts. The condition is differentiated from acanthosis nigricans by the discreteness of the papillary hypertrophy and by the presence of atrophy. The cause could not be determined. F. F. H.

**PEMPHIGUS AND PEMPHIGOIDS.** A. Touraine. (1954) *Ann. Derm. Syph., Paris*, **81**, 121.

The author divides bullous eruptions into the above two groups ; in the former are the classical forms of pemphigus whilst the latter includes traumatic blisters, allergic reactions, infections, congenital lesions, dermatitis herpetiformis, etc. The author shows that there is no clinical chemical or haematological method of distinguishing dermatitis herpetiformis from pemphigus. Histology appears to give the only clear separation but he quotes various authors who do not accept the classical findings of Civatte. The so-called bullous pemphigoid is only briefly mentioned. The paper contains an interesting discussion on diagnosis and aetiology and a full bibliography. F. F. H.

**OCCUPATIONAL DERMATITIS.** Bentley Phillips. (1954) *The Practitioner*, **172**, 531.

This paper is based on an investigation carried out on 250 consecutive cases of occupational dermatitis, 150 males and 100 females, with a control group of 100 adult hospital out-patient non-compensation cases, suffering from contact dermatitis due to substances encountered at home, i.e. household or garden irritants. An interesting feature of this paper is that, in the control group due to household or garden irritants, 45% of the 100 patients admitted to a previous history of asthma, hay fever, eczema or urticaria either in themselves or in their families, whereas in the compensation cases only one patient out of the 250 admitted to a personal history of asthma and one to a family history of asthma. From this, the author concludes that where compensation is in question, a history of the absence of previous allergic troubles is completely unreliable. He found that a large proportion of the compensation cases had either a seborrhoeic or an eczematous diathesis and that, by greater efforts at personnel selection, a great deal of occupational dermatitis could be avoided. Out of the 139 cases followed up for periods between one and four years, every one was still being paid compensation—not one had fully recovered and been able to resume

his original occupation without further troubles; and "hardening" had not been in evidence. The veracity of these patients is in doubt as, without exception, all the housewives appeared to live lives of complete leisure! A. C. R.

**SEBORRHOEIC DERMATITIS.** A. Rook. (1954) *The Practitioner*, **172**, 522.

The author thinks that the composition of sebum is of greater clinical significance than the quantity secreted. He considers that the rôle of pityrosporon ovale is still debatable. In summary, seborrhoeic dermatitis is probably a diffuse infection of a pathologically altered horny layer, the variations in the clinical picture depending upon the relative predominance of streptococcal, staphylococcal or other infection and the severity and character of the underlying disturbance of cutaneous function. He considers seborrhoeic dermatitis in its usual forms and discusses the various types of treatment. A. C. R.

**SKIN DISORDERS CAUSED BY PYOGENIC ORGANISMS.** P. J. Hare. (1954) *The Practitioner*, **172**, 516.

The author considers the common pyogenic diseases of the skin. In treatment, he thinks that soaps and shampoos containing hexachlorophene seem more promising than the more old-fashioned antiseptic soaps. In follicular infections, he has found the substituted quinoline compounds very useful. As regards antibiotics by local application, he has found aureomycin particularly useful in chronic follicular infections; while oxytetracycline, neomycin and bacitracin and combinations of the latter with polymyxin and tyrothricin appear to be effective and to provoke little contact dermatitis, the majority have also the advantage that they are unsuitable for systemic administration. Chloramphenicol causes little contact dermatitis but is not always effective against skin staphylococci. On the other hand, streptomycin very readily causes severe contact dermatitis and should not be used locally on the skin. A. C. R.

**THE SKIN IN OLD AGE.** G. H. Percival. (1954) *The Practitioner*, **172**, 510

The author considers the changes in the skin with age and their relation to infections, allergic manifestations, other skin diseases, atrophy, hypertrophy and carcinoma. A. C. R.

**CORTISONE AND ACTH IN DERMATOLOGY.** F. F. Hellier. (1954) *The Practitioner*, **172**, 503.

The author is in a particularly strong position to review this aspect of cortisone and ACTH therapy because he is secretary of the Cortisone and ACTH dermatology panel of the Medical Research Council. He thinks that hydrocortisone ointment, not yet available in this country, is clearly a therapeutic agent of considerable significance. He divides the skin conditions in which the use of cortisone and ACTH must be considered into four groups: (1) fatal or severely incapacitating conditions, in which these substances may be the only controlling treatment available; these include pemphigus, acute lupus erythematosus, generalized exfoliative dermatitis and polyarteritis nodosa; (2) severe self-limiting conditions in which cortisone and ACTH produce marked improvement and may greatly diminish the patients' suffering, such as drug reactions, severe dermatitis venenata, Stevens-Johnson syndrome and dermatomyositis; (3) chronic non-fatal skin diseases, such as Besnier's prurigo, psoriasis, lichen planus, urticaria, alopecia, and pityriasis rubra pilaris. As a whole, the author does not consider that cortisone and ACTH are ordinarily justifiable in these conditions; (4) chronic serious dermatoses, loosely labelled reticuloses, mycosis fungoides, Hodgkin's disease and leukaemia. Cortisone and ACTH have no real effect on the course of these diseases. After describing the toxic reactions liable to occur in treatment with cortisone and ACTH, the author concludes that, with such serious potential dangers, it is obvious that the advantages and disadvantages of cortisone and ACTH treatment must be carefully weighed for each individual patient. A. C. R.

**THE SKIN AND THE BODY.** I. B. Sneddon. (1954) *The Practitioner*, **172**, 493.

The author considers a number of internal diseases that are associated with changes in the skin, i.e. erythema multiforme, erythema nodosum, guttate psoriasis, glandular fever, drug rashes, lupus erythematosus and polyarteritis nodosa, blood dyscrasias and the reticuloses.

A. C. R.

**WARTS.** E. W. Prosser Thomas. (1954) *The Practitioner*, **172**, 538.

The author discusses warts in their different varieties and describes the appropriate treatment for them, coming to the depressing conclusion that it must be admitted that no method of treatment for warts is entirely reliable and it can never be guaranteed that there will be no recurrence.

A. C. R.

**OBSERVATIONS ON AUTOSENSITISATION IN CONTACT DERMATITIS.** S. Epstein. (1953) *J. invest. Derm.*, **21**, 183.

In the few cases at his disposal the author has made the interesting observation that serum, from a subject who was or who in the past had been chrome-sensitive and who had recently been patch tested (whether the reactions were positive or not), injected intradermally into another chrome-sensitive subject gave a tuberculin type reaction. Serum taken before or a week after the patch test would not work.

This reaction could not be elicited in the case of nickel. But the serum of a rhus-sensitive subject who had recently received a therapeutic injection of extract gave a positive reaction while that of another rhus-sensitive who was in the middle of an attack of chrome dermatitis did not.

These results enable the author to speculate rather freely. Does patch testing stimulate the production of a secondary antigen ? Then why doesn't it need to be positive ? Is it because there are 2 kinds of antibody, a sensitizing one and a neutralizing one ? If so we have no difficulty in explaining, if indeed there was ever a need to explain, why the case of acute rhus dermatitis did not react to his own serum. But let Occam sharpen his razor. J. H. T. D.

**A CHEMICAL MEASURE OF THE EFFECT OF SOAPS AND DETERGENTS ON THE SKIN.** E. J. van Scott and J. B. Lyon. (1953) *J. invest. Derm.*, **21**, 199.

There was considerable variation among 15 detergents in their capacity to expose –SH groups in keratin : and a clinical investigation is already planned for the correlation of this chemical reaction with the incidence of dermatitis.

While one can easily see that such a reaction might well be associated with visible changes in the horny layer, the connection with " dermatitis " is not so easily grasped. While one very large institution is telling us that its employees have used 1000 tons of various detergents without a single case of dermatitis, a few miles away the disease may form the main topic of conversation at a vicarage tea party.

That the diagnosis of " housewife's dermatitis " is often made in Philadelphia is implied in the opening paragraph, which at the same time reminds us that we do not know yet whether it is the defatting action, the pH, direct chemical action on the skin, " sensitizing potential " or merely superstition that does it.

Anyhow the authors promise to tell us, in their next, exactly what happens to the keratin molecule—which will be something to be going on with. J. H. T. D.

**THE COURSE OF TRICHOPHYTIN GYPSEUM INJECTION IN RABBITS PREVIOUSLY INJECTED WITH TRICHOPHYTIN PURPUREUM. THE EFFECT OF ACTH ON THE TRICHOPHYTIN TEST.** F. Reiss, E. M. Rosenbaum and Leona Caroline. (1953) *J. invest. Derm.*, **21**, 191.

Twenty rabbits were used in the main part of the experiment. In each of them 6 sq. in. of skin was abraded with knife or sandpaper and a culture preparation of *T. purpureum* rubbed in. A crusted lesion, not here fully described, developed in which the fungus survived at least 10 but not much more than 30 days. On repeating the process a reaction was obtained which in no way differed from the first. After 3 months the same rabbits were inoculated in the same fashion with *T. gypseum*. Exactly the same effects were observed in the castrated rabbits that had previously been " infected " with *T. purpureum* as in 3 entire rabbits without previous contact with ringworm fungus.

Although chronic infection with *T. gypseum* was not obtained either in castrated or entire rabbits nor with *purpureum* in castrated ones, there does not seem to be much evidence of the phenomena we ordinarily associate with immunity.

Next the rabbits are injected with a particular brand of trichophytin which was found to give positive reactions in 14 out of 19, presumably the castrated rabbits that survived the initial inoculation with *T. purpureum*. The other brands of trichophytin do not seem to have worked so well, but the authors do not say whether any of them were tried on uninfected controls.

Finally the rabbits are given 6.25 mg. per kilo of ACTHAR (by which 2 die and 2 more are damaged) and the trichophytin reaction—contrary to the experience of other investigators—is suppressed.

The length of this abstract is less an indication of the importance of the paper than of the need for translation. J. H. T. D.

**TUBERCULOSIS OF THE SKIN.** C. Sobanadri, M. V. Krishnamurthi and D. J. Reddi. (1954) *Antiseptic*, **51**, 684.

Twenty-three cases of lupus vulgaris are described; only two occurred on the face, most being on the neck or upper trunk, possibly a reflection of the less clothing needed in India. Remarkably short courses of streptomycin, with or without P.A.S. depending on whether or not pulmonary disease was present, are claimed to have produced highly satisfactory results, which are recorded in large numbers of " before and after " photographs. The relapse rate may prove disappointingly high.

In expressing their hopes of still better things to come from the exhibition of isoniazid, the authors refer somewhat anagrammatically to the report on this drug in lupus by the President of the Royal College of Physicians. R. D. S.

**OBSERVATIONS ON POLYDACTYLISM.** W. F. Harper. (1954) *W. Ind. med. J.*, **3**, 12.

Small post-axial rudiments may occur arising from the base of a little finger or toe. This type is often familial. A partially developed digit may articulate with the side or head of a metatarsal (carpal) which already has its own proper digit, or a digit may be partly or completely bifurcated.

Lastly, like the giant mentioned in 2 Sam. xxi, 20, with " on every hand six fingers and on every foot six toes, four and twenty in number " the lucky person may have more than his quota of fully formed digits. The author describes and illustrates two male cases of the latter variety, a negro baby with seven toes on his left foot, fully formed with their own metatarsals and cuneiforms and a fair supply of muscle attachments, and a white child with six left and seven right toes. In the first the two medial toes were the extras, having three phalanges, and were removed, cuneiforms and all. In the second case the second were the odd ones and also the sixth on the left and again they had their own metatarsals though the tarsi were normal. These also were removed. R. D. S.

**PEMPHIGUS.** V. Sridhara Rao. (1954) *Antiseptic*, **51**, 794.

A good general and up-to-date account of the various types of pemphigus and their treatment. Bullous pemphigoid is included as a variant, and the first of three cases described seems to be an example of this rather than of true pemphigus. R. D. S.

**THE EFFECT OF SWEAT ON THE QUANTITY OF ETHER SOLUBLE SUBSTANCES ON THE SKIN.** F. Herrmann, P. H. Prose and M. B. Sulzberger. (1953) *J. invest. Derm.*, 21, 397.

Lipoid is a regular product of cornification, and apparently it is stored within and between the cells of the horny layer. But it must be doubted if enough could be secreted in this way to account for the "replacement sums " here recorded for palmar surfaces. But it also seems certain that the covering epithelium secretes lipoid at the same time as sweating takes place and that the presence of sweat on the surface attracts and emulsifies lipoids both from the horny layer and sebaceous gland.

In these experiments the attempt is made to demonstrate both physiological and chemical interdependence of sweat with the lipoid of the horny layer. Measurements of ether soluble materials, and of the blackening of the surface by osmic acid vapour are applied to the comparison of skin treated with atropine and pilocarpine. Studies of the mutual emulsification of lipoid and sweat are pursued *in situ* and *in vitro*.

The paper is not an easy one to read, but the fault is not with the authors so much as with a subject that offers little more than unimpressive differences and hints of inscrutable mechanisms. For that it is all the more welcome. J. H. T. D.

**STUDIES ON THE ACTION OF PARAPHENYLENE DIAMINE ON LIVING CELLS.** A. G. Matoltsky. (1953) *J. invest. Derm.*, **21**, 447.

It is the oxidation products of paraphenylene diamine that are toxic and also, perhaps, responsible for the sensitization phenomena. For patch testing one should therefore use the base rather than the dihydrochloride which is well protected against oxidation and may give false negative reactions. In these experiments tissue cultures of chicken heart were treated with paraphenylene diamine. The addition of a reducing agent, sodium sulphite, greatly diminished the effect which, short of complete inhibition of growth, retarded it and accelerated fatty degeneration. J. H. T. D.

**COMPARISON OF SKIN COLOR WITH MELANIN CONTENT.** R. R. Gates and A. A. Zimmermann. (1953) *J. invest. Derm.*, **21**, 339.

White races differ from black only in the tyrosinase-inhibiting SH groups. They have just as many dendritic cells or melanocytes, the function of which is to produce the pigment and transfer it to other basal and rete cells.

Save in very dark and very pale skins the pigment is unevenly distributed ; nevertheless it is not difficult to correlate unstained sections with complexion. The author refers to yet unpublished work in which he has developed the theory that the degree of pigmentation of an individual is related to his pedigree. J. H. T. D.

**ON THE MECHANISM OF MELANIN PIGMENTATION IN ENDOCRINE DISORDERS.** A. B. Lerner, K. Shizume and T. B. Fitzpatrick. (1953) *J. invest. Derm.*, **21**, 337.

Any pituitary extract will blacken frogs, but only those containing a satisfactory amount of M.S.H. ("melanocyte stimulating hormone", " melanophore hormone", " melanophore dilating principle", " intermedin", etc.) are able to produce a healthy tan in man. Such an extract is obtained from the pituitary of hogs—presumably black ones—but is also found in the urine of pregnant women and people with Addison's disease. It is therefore presumed that the pigmentation in these conditions is brought about by M.S.H.

The metabolism of M.S.H. is controlled by antagonists. Adrenalin inhibits the local action of M.S.H. on the melanocytes. Hydrocortisone inhibits its release from the pituitary. Progesterone has an independent M.S.H.-like action. J. H. T. D.

**PEMPHIGUS AND OTHER BULLOUS DERMATOSES : CORRELATION OF CLINICAL AND PATHOLOGIC FINDINGS.** J. G. Brennan and H. Montgomery. (1953) *J. invest. Derm.*, **21**, 349.

Montgomery who in 1927 " ... followed the concept that Gans, Unna, Hebra and others had that any bullae without eosinophils or any cells in the bullae were pemphigus and otherwise not ... " now believes with Brennan that the true criterion is acantholysis and adduces convincing proof of it. The work is a clinical and histological study of " ... patients who had serious bullous dermatoses encountered at the Mayo Clinic since 1907. Special emphasis was placed on those in whom the diagnosis of pemphigus in any of its forms was suspected". This seems to mean that the study concerns cases in which any diagnosis was made that contained the word pemphigus.

A useful table indicates the proportions of the various conditions called pemphigus in a total of 508 cases, together with age, sex and race incidence. J. H. T. D.

**THE INFLUENCE OF HYALURONIC ACID AND OTHER SUBSTANCES ON TENSILE STRENGTH OF HEALING WOUNDS.** H. Pinkus and E. T. Perry. (1953) *J. invest. Derm.*, **21**, 365.

An incision was made on each side of the midline of the back in guinea-pigs and allowed to heal. The substances to be compared were either introduced through tiny tubes left in the depth of the wound or were injected subcutaneously twice a week. Groups of 6 were " sacrificed " at 5-day intervals between the 15th and 35th day after operation.

Tensile strength was measured by inflating the skin with air under water (to detect the leak). The histology of the events was disappointingly unilluminating.

It seems that hyaluronic acid, though it may seem to interfere with the earliest stages of healing, can in the end bring about a firmer union. J. H. T. D.

**TIME-DOSAGE RELATIONSHIP IN THE TREATMENT OF TREPONEMAL DISEASES WITH A NEW COMBINATION OF 3 PENICILLIN SALTS. LABORATORY AND CLINICAL BASIS FOR EFFECTIVE THERAPY.** C. R. Rein, F. H. Buckwalter, C. H. Mann, S. E. Landy and S. Flax. (1953). *J. invest. Derm.*, **21**, 435.

Five injections of this mixture at daily intervals create immediate blood levels of 4 units per c.c., falling to 0·4 24 hours after the last injection, but persisting at 0·1 for as long as 3 weeks.
J. H. T. D.

**STUDIES IN THE MODE OF ACTION OF VITAMIN A.** P. Flesch. (1953) *J. invest. Derm.*, **21**, 421.

*In vitro* vitamin A prevents keratinization of tissue-cultured epidermis ; it produces reversible baldness both when applied to the skin and when ingested. It also has a slight improving effect, probably not much better than salicylic acid, on ichthyosis. These effects are here shown to be pharmacological, due to a particular chemical configuration shared with other materials that are not vitamins. Vitamin A is not to be found in the skin. J. H. T. D.

**EPIDERMAL PROTEASE.** G. C. Wells and C. Babcock. (1953) *J. invest. Derm.*, **21**, 459.

Beloff and Peters (1945) described a proteolytic enzyme in the skin of animals. The authors have now established its epidermal origin, and they have further described it. They speculate on its possible function in the healing of wounds. J. H. T. D.

**LUPUS ERYTHEMATOSUS AND POLYMORPHOUS LIGHT ERUPTIONS. AN EXPERIMENTAL STUDY ON THEIR POSSIBLE RELATIONSHIP.** M. M. Cahn, E. J. Levy, B. Shaffer and H. Beerman. (1953) *J. invest. Derm.*, **21**, 375.

Of 17 patients having polymorphous light eruptions, some of them of the prurigo aestivalis type others more suggestive of lupus erythematosus, only 2 reacted (in the neighbourhood of the " V " of neck) with specific papules to a 12-times erythema-dose of cold quartz light. A 5 times erythema-dose gave only a banal reaction. The authors comment on this well-known difficulty of obtaining a specific reaction to light at will and its spontaneous occurrence often with minimal exposure but they fail to discuss the possibility that the noxa may appear only intermittently in the circulation. The histology of specific papules both natural and artificial resembled, in a significantly great number of respects, that of lupus erythematosus.
J. H. T. D.

**THE EFFECT OF HYDROCORTISONE ACETATE OINTMENT ON PRURITUS ANI.** R. M. Alexander and S. D. Manhein. (1953) *J. invest. Derm.*, **21**, 223.

In 25 of 29 patients not only did the symptom disappear but the skin is said to have returned to normal. In the absence of any statement to the contrary this is taken to mean that the anus became repigmented and the folds resumed their normal pattern. But only 3 of the patients have been able to discontinue treatment. Since continued therapy may be required it is particularly satisfactory to know that the ointment has an agreeable smell. J. H. T. D.

**DERMATOLOGY OF TO-MORROW.** T. Cornbleet. (1953) *J. invest. Derm.*, **21**, 215.

To avoid extinction of his speciality the dermatologist must to an increasing extent justify his existence as a scientist. Since antibiotics and hormones have brought many important therapeutic problems within the capacity of the unlearned, and so long as the cutaneous manifestations of psychoneurosis remain the prerogative of the charlatan, there is not much left for him to do beyond description, research and teaching. In order that he shall not teach what is merely charlatanry he must have behind him at least one scientific discipline whereby he personally can maintain a fruitful pressure against the boundaries of his science.

The writer welcomes the increasing participation of dermatologists in investigative studies. He reviews new knowledge of the connective tissue ground substance, the isotope tracing of metabolic intermediates and the new conception of biological antagonists. He indicates several questions that still wait to be answered. J. H. T. D.

**THE *IN VITRO* ANTIMICROBIAL ACTIVITIES OF CERTAIN AMINOSTEROIDS.** F. C. Kull, G. A. Castellano and R. L. Mayer. (1953) *J. invest. Derm.*, **21**, 227.

Six steroid hormones from the shelves of a pharmaceutical manufacturer exhibited antibacterial and antifungal properties.
J. H. T. D.